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Section of the History of Medicine

President—Sir STCLAIR THOMSON, M.D.

[*May* 2, 1934]

The History of Ovariotomy

By HERBERT R. SPENCER, M.D.

THE hybrid word *ovariotomy* was first used by Charles Clay of Manchester as a caption in his book *Peritoneal Section for the Extirpation of Diseased Ovaria*, published in 1848 [1]. It is not accurately descriptive of that operation, any more than is its modern substitute *ovariectomy*; but from a historical standpoint the term may be used to indicate the removal of an ovarian (or parovarian) tumour, including the excision of a tumour from the ovary, with retention of the rest of the organ.

It has been much disputed who was the first to perform the operation of ovariotomy. In 1897 there was a lively discussion between Greig Smith who denied and Lawson Tait who asserted the claim of Robert Houstoun, as having performed the first ovariotomy, in the year 1701. What the record [2] states is that Houstoun removed a large quantity of fluid and glutinous material, together with some "hydatids" through an abdominal incision, and that the patient survived the operation for thirteen years. Houstoun makes no mention of a pedicle, the absence of which, as Tait remarks, does not preclude the possibility of the tumour's being ovarian; but neither does it preclude the possibility that the material originated in pseudomyxoma peritonei arising from the vermiform appendix or other abdominal organ. While therefore it must be admitted that Lawson Tait, a master of dialectic, makes out a good case for Robert Houstoun as *possibly* the pioneer in ovariotomy, it was more than a century later than the date of his operation that the first undoubted ovariotomy was performed by Ephraim McDowell of Danville, Kentucky, in the year 1809. The heroic Mrs. Crawford, the patient on whom he operated, thought that she was pregnant and beyond her term. She was seen by McDowell who diagnosed an ovarian tumour, which he thought it was possible to remove, although the operation was of the nature of an experiment. The patient promptly accepted the proffered aid and soon afterwards made a journey of sixty miles on horseback to Danville, where the tumour was removed by McDowell, the patient making an excellent recovery. Subsequently McDowell performed two successful ovariotomies and sent an account of them to his old teacher John Bell, of Edinburgh, which came into the hands of Lizars (Bell's locum tenens) who performed the first ovariotomy in Great Britain, in 1825. The account of McDowell's three cases appeared in print in the *Eclectic Repertory and Analytical Review*, published in Philadelphia in October 1816, but in such a brief form that writers were incredulous and sarcastic, one editor openly expressing his disbelief in McDowell's statements; a few years later, however, he "acknowledged his error and for his uncharitableness asked pardon of God and Dr. McDowell." The abdominal incision was made by

McDowell externally to the border of the rectus or, later, in the linea alba. The pedicle was ligated before opening the cyst and the peritoneum emptied of fluid by turning the patient on her side. Drainage was provided by bringing the ligature out through the lower end of the incision, from which the ligature was withdrawn by traction or spontaneously separated after about five weeks.

In all McDowell performed thirteen ovariotomies, with eight recoveries and five deaths (38%).

He was a very successful operator in other branches of surgery, especially in lithotomy, which he performed in 22 cases without a death : one of these was on a boy of fourteen, J. K. Polk, who afterwards became President of the United States. McDowell married the daughter of the Governor of Kentucky. He died on June 20, 1830, in his fifty-ninth year.

To perpetuate the name and fame of this heroic pioneer a monument was erected over his grave at Danville by the Kentucky State Medical Society on May 14, 1879, and on April 22, 1909, the American Gynecological Society celebrated the Centenary of Ovariotomy by a meeting and banquet at the Waldorf-Astoria Hotel, New York, at which addresses on McDowell and Mrs. Crawford were delivered by Dr. McMurtry and Dr. E. P. Davis, and on McDowell's successors in America by Dr. Howard Kelly, in Great Britain by Dr. Herbert R. Spencer, in France by Professor Pozzi, and in Germany by Professor Hofmeier. A bronze medal, struck for the occasion, was presented to the Fellows, and relics from the house of McDowell were exhibited : two of these had been made into gavels for the use of the chairman at the Society's meetings [3].

After McDowell's operation in 1809, the next American ovariotomist was Nathan Smith, who, in 1821, successfully removed an ovarian cyst through a subumbilical incision, tied the arteries of the pedicle with ligatures made from a kid glove, cut the ends of the ligatures short, dropped the pedicle and closed the wound by adhesive strips, no suture being applied to the abdominal wound.

Alban Smith, of Danville, the partner of McDowell, did the the third successful ovariotomy in America, in 1823, tying the pedicle with strong silk, the ends of which were brought out of the lower end of the wound. D. L. Rogers, of New York, in 1829, and J. Billinger, in 1835, operated successfully, tying the vessels separately (Billinger using animal ligatures) and cutting the ends short. Other celebrated ovariotomists were J. L. Atlee (who operated in 1843) and continued to operate for forty years (78 cases with 64 recoveries) and W. L. Atlee, who performed his first successful operation in 1844, and is said by Howard Kelly to have done his last ovariotomy in 1878, bringing his list up to 387 cases, with a mortality rate of "about 30%." Dunlap operated first in 1843 ; the operation was unsuccessful and he was blamed for doing "an unjustifiable and murderous operation" ; he operated successfully in 1849. In 1850 Peaslee did his first operation and is said by his son to have performed in all 78 ovariotomies, with "about 60 recoveries."

After the publication of W. L. Atlee's 35 cases, in 1855, opposition to the operation lessened, and a doctor who had called Peaslee "the greatest quack in Philadelphia" was, within a year, following in Atlee's footsteps. Peaslee used the short incision and leather ligatures, cut short and dropped into the peritoneum.

Ovariotomy in France.—In France the performance of ovariotomy was delayed by the opposition of the great surgeons Dupuytren, Boyer, Giraldès, Velpeau [4] and the Academy of Medicine which condemned it in spite of the protest of Cazeaux. Up to the year 1860 only two ovariotomies had been done in France, both successfully by provincial surgeons, Woycikowski (April 28, 1844) and Vaullegard (September 15, 1847). In 1861 Professor Nélaton came to England to see Baker Brown operate, and on February 2, 1862, Demarquay operated on a girl aged 19, whom he had removed from the unhealthy surroundings of the Paris Hospitals to Saint Germain. The patient died on the third day from peritonitis. In the same

year Koeberle of Strasbourg began his career as an ovariotomist by performing a successful operation on June 2 and two others successfully in the same year. Up to 1878 he performed 293 cases with 75 deaths (25%). His Paris confrère Péan, up to the end of 1887, performed 638 ovariotomies with 93 deaths (14·5%). Péan, or Koeberle, was the inventor of the hæmostatic forceps (slightly altered by Spencer Wells), and these surgeons (like Spencer Wells even as late as 1878, when he adopted the ligature) treated the pedicle extraperitoneally by means of a clamp, the invention of Hutchinson in 1858. Koeberle devised a very useful *serre-noeud* for the purpose of constricting the pedicle, which proved a valuable means of treating the uterine stump extraperitoneally in the operation of hysterectomy. Terrier began to practise the operation in 1877, using no antiseptics, but paying great attention to cleanliness. From 1878 he adopted the Listerian method. He tied the pedicle with catgut or, later, carbolized silk, cut short and dropped. Up to July 1889, he had done 200 ovariotomies, with 37 deaths (18·5%). In the year 1886 Pozzi [5] introduced the suture of the abdominal wound in layers, now adopted by all, and the endermic suture of the skin, which he afterwards abandoned as a routine practice, owing to the loss of time it entailed. He also used the aseptic method of operating in 1890, which was followed by Terrier and his pupils, and has, since the end of last century, been universally employed.

Ovariotomy in Germany.—In Germany the first ovariotomy was performed by Chrysmar, a general practitioner at Isny in Würtemberg, in the year 1819. He made a large incision, separated the adhesions, ligatured the pedicle and brought the ligatures out of the lower end of the wound. The patient died of peritonitis on the second day. In 1837 Stilling, of Cassel, in performing the operation fixed the pedicle in the abdominal wall, with the object of avoiding bleeding and suppuration. He also suggested the extraperitoneal treatment of the pedicle in 1841, and carried out the suggestion in 1848. This method was afterwards adopted by E. Martin, Langenbeck, Kiwisch, Scanzoni and Bardeleben; but the results were generally very bad (Hofmeier) until the German surgeons had visited England to see the work of Clay, Baker Brown, Keith and Spencer Wells. Schroeder was the first German ovariotomist to adopt Listerism, in 1878. Hofmeier states that Schroeder performed a larger number of ovariotomies than any other German, 658 in all, with 83 deaths (12·6%). He used to suture the incision with silk sutures passed through the whole thickness of the abdominal wall.

Ovariotomy in Great Britain.—At the end of the eighteenth century John Hunter and William Hunter, and at the beginning of the nineteeth century, John Bell, had foreshadowed the possibility of the removal of ovarian tumours, but it was not till 1823 that any scientific work was done in this connexion, when Dr. James Blundell, lecturer on midwifery at Guy's Hospital, submitted to the Royal Medical and Chirurgical Society a paper on abdominal surgery, based on experiments which he had made on living animals. The paper was read before the Society, but was not accepted for publication; it was however, published by the author in the same year. It contained reports of scientific experiments on ovariotomy and hysterectomy which may still be read with interest and profit; guided by these he was the first to remove the cancerous uterus in this country. But the paper was vigorously assailed by the vivisectionists, to whom Blundell appealed to "strike, gentleman, but hear! you who would join the laugh at the Egyptians, which will you sacrifice, your women or your cats?" Of ovariotomy Blundell writes, "This operation will, I am persuaded, ultimately come into general use." He lived to see the fulfilment of his prophecy and died in 1878 at the age of 87, leaving a fortune of £350,000.

Lizars, of Edinburgh, was the first to perform ovariotomy in Great Britain, in 1825. He acknowledged that the operation he performed was founded on the general principles contained in Blundell's paper, but he was probably influenced also by McDowell's account of his own operation, which McDowell had sent to his master,

John Bell, about the year 1817, and came into Lizars' hands when he was acting as *locum tenens* for Bell who had gone abroad on account of illness.

During the next fourteen years the progress of ovariotomy was slow, at the rate of one operation a year, and was entirely confined to the provinces until the year 1839, when, probably as a result of Blundell's influence, it was introduced for the first time into a metropolitan hospital, Guy's, where it was performed by Morgan, Key and Bransby Cooper. In the provinces successful operations had been done by Jeaffreson of Framlingham and by King of Saxmundham and West of Tonbridge in 1836; West had a second successful case in 1839. In 1842 Charles Clay, of Manchester, performed the operation successfully in his first three cases and for many years he was the chief ovariotomist in Great Britain, to whose exertions the revival of the operation was chiefly due. It is to him that we owe the word *ovariotomy* suggested to him by Sir James Simpson, who, writing in 1860, said that Clay had performed about one-fourth of the ovariotomies done up to that time. In the same year John Clay [6] published statistics of the ovariotomies up to that year, 212 in all, with 183 deaths (86%): of these 65 completed operations had been performed by Charles Clay with 29 deaths (44%), 10 by Spencer Wells with seven deaths (70%), and 10 by Atlee with nine deaths (90%).

Charles Clay's first case was operated on June 20, 1842. Clay used the long ligature brought out of the wound, and is said by his fellow-townsman, the late Dr. Lloyd Roberts, to have performed, in all, 395 ovariotomies with 101 deaths (25·5%). In 1848 Spencer Wells, who was to become our greatest ovariotomist, came to the conclusion that ovariotomy was not a justifiable operation, a conclusion to which the greatest British obstetrician of the latter half of the nineteenth century, Matthews Duncan, also arrived, alleging that the defenders of ovariotomy had nothing but flimsy and fallacious arguments to advance in its support.

In 1846 Burd, of Salisbury, performed the first ovariotomy during pregnancy, adopting the useful practice of separate ligature of the vessels.

In 1851 Baker Brown began his brilliant career as an ovariotomist; up to 1867 he had operated on 111 cases with 35 deaths (31%). He cauterized and dropped the pedicle and attributed his good results to that method of treatment, which was certainly superior to the clamp and the long ligature.

In 1854 Spencer Wells and T. Nunn assisted Baker Brown in his eighth ovariotomy. This was the first time Spencer Wells had seen the operation performed. Four years later, in 1858, Spencer Wells performed his first ovariotomy and began that remarkable career which placed him for many years in the position of the world's greatest ovariotomist. By his steadfast plan of publishing all his cases he was finally able to break down all opposition to the operation, and to his success was largely due the general extension of abdominal surgery.

Surpassed by several of his contemporaries in technical skill, disinclined to accept improvements introduced by his rivals, e.g. the cautery introduced by Baker Brown, the short ligature introduced (after the Americans had used it) by Tyler Smith, he possessed the qualities of doggedness and grit which enabled him, with his strong personality, to force the operation on the profession, which will ever owe him a great debt. In 1880 Spencer Wells published his "1,000 Cases of Ovariotomy," in which the mortality rate was 23·1%. In 1865 Lister introduced his antiseptic treatment for compound fracture and in 1877 came to London where, in the following year (1878) the Listerian system was adopted in ovariotomy by Spencer Wells, Thornton, Keith and John Williams (who was the first to perform ovariotomy during labour). Before the adoption of the antiseptic system the rate of mortality was high. Thomas Keith, who began to operate in 1862, was the most successful operator, but he lost 11 of his first 50 cases; he cauterized the pedicle and employed a glass drainage-tube. Thornton lost seven of his first 25 cases; Lawson Tait 19 of his first 50. After the adoption of the antiseptic system Thornton lost only three

of his next 50 cases, and generally the mortality rate was still further reduced by those using the antiseptic method and its modification, the aseptic treatment, universally practised since the end of last century.

Two of the most successful ovariotomists, Lawson Tait (who introduced the Staffordshire knot to tie the pedicle, and the practice of flushing the peritoneum) and Bantock did not attribute their good results to the use of antiseptics, but rather to cleanliness in the ordinary sense; but they do not appear to have entirely dispensed with the use of antiseptics. They were certainly among the first to recognize the powers of the peritoneum in dealing with mild infections, and had a considerable influence in promoting the aseptic method.

Ovariotomy in Italy.—The first to perform the operation in Italy was G. Emiliani in the year 1815, who removed a tumour which was probably a soft fibroma; the next was Vanzetti, in 1859. Thus in Italy, as in France, only two ovariotomies had been done before 1860. Since that date the Italian gynæcologists have made up for their early abstention by the excellence of their contributions. Amongst others, F. Vozza [7] has published, from the clinic at Milan, the largest list which has yet appeared of operations for ovarian and parovarian tumours complicating pregnancy (109 cases, with three deaths).

Ovariotomy in other countries.—In Denmark the first ovariotomy was done in 1863; it was unsuccessful, as were all those performed up to the year 1867, when Boye did the first successful one; in all he did 70 ovariotomies, with a mortality rate of 40%.

In Sweden, at about the same time, Reisz had performed three unsuccessful ovariotomies at Stockholm, and Mesterton three successful ones at Upsala.

In Russia the first ovariotomy performed by a native of that country was Krassowsky's in 1862.

In Spain, from 1863 to 1885 there were 61 cases, with 38 deaths (55%).

In China the first successful operation was done by Kerr in 1880.

I have not been able to discover who performed the first ovariotomy in Japan. In 1892 Omori and Ikeda published a list of 150 ovariotomies, of which in no less than 36 the tumour was a dermoid. Amongst my own 658 ovariotomies 66 were for dermoids (about 10%) which agrees closely with the general average (10·94%) [9]. Yamasaki [8] has shown that amongst 1,408 ovariotomies performed by nine Japanese gynæcologists there were 319 dermoids (22·65%), Yamasaki himself having met with 71 dermoids (34·97%) amongst 203 ovariotomies he had performed. The high frequency of dermoids in these Japanese series is so much greater than that usually met with as to suggest that it is possibly due to racial idiosyncrasy.

In India ovariotomy was performed successfully by a native in 1860, and unsuccessfully by K. McLeod in 1868. In Calcutta, since 1878, 42 ovariotomies were performed by Harvey (mortality rate 23·74%); 75 by Joubert and Peck (mortality rate 18·5%); 574 by Green-Armytage (mortality rate 7·4%).

Mortality of ovariotomy.—The immediate mortality of the operation has naturally fallen below that of the early pioneers, who had not the advantages conferred by anæsthesia and antiseptics and the experience of others. It varies with the operative material (malignant, suppurating, ruptured, twisted, adherent tumours) and with the age of the patients. In Spencer Wells' 1,000 cases only two patients were over 70 years of age, only 12 of the cysts ruptured, and three suppurating: in my own series of 658 cases [14] there were 11 patients over 70 years of age, 41 ruptured, 53 suppurating and 67 malignant tumours. The recorded mortality-rate varies also with the time the patient remains under observation, whether fifteen days, as is the custom with some operators, or twenty-four days, as is my practice. Thus, had I adopted the practice of the earlier date of discharge, my mortality rate would have been understated at 4·2 instead of 5·3%.

A. Mayer, in a collected series of modern cases, found the mortality rate to be

5·7%, which I think is a fair average at the present time, if no selection of cases be made. The following is a list of the results obtained by gynæcologists who have personally performed at least 500 ovariotomies :—

	Number	Percentage mortality
Spencer Wells [10]	1,000 ...	23·1
Péan [11]	638 ...	14·5
Hofmeier [12]	500 ...	11·6
Schroeder [13]	658 ...	12·6
H. R. Spencer [14]	658 ...	5·3
Green-Armytage [15] ...	547 ...	7·4
Lawson Tait [16]	677 ...	6·0

The abdominal incision :—

The incision in the abdominal wall has varied in the course of time, having been longitudinal, long or short, or (since the end of last century) transverse through the skin and fat (Küstner), or through skin, fat and fascia (Pfannenstiel). McDowell made his long incision to the left of the rectus muscle in the case of Mrs. Crawford, who had bruised her abdomen against the pommel of the saddle in her long ride on horseback: in later cases he incised in the linea alba, using the long incision, which is still in favour, as it allows moderate-sized tumours to be removed whole, thus avoiding infection or implantation from the contents. Longitudinal incisions are also made through the inner edge of the rectus, and through the anterior sheath, opening the posterior sheath and peritoneum behind the muscle, after retracting the muscle outwards, with the object of lessening the risk of hernia of the scar. This risk is minimized by the transverse Pfannenstiel incision, which is excellent for the removal of small tumours, especially those complicating early pregnancy.

In order to diminish the bulk and facilitate the extraction of large cystic tumours Spencer Wells invented an ingenious, but not always effective, trocar; and Lawson Tait used a simpler cutting-tube for the purpose. A device, proof against leakage, is still a desideratum.

Vaginal ovariotomy.—Owing to the general lower mortality of vaginal than of abdominal operations, and to avoid an abdominal scar and a subsequent hernia, vaginal ovariotomy has been resorted to by many gynæcologists. It was first performed in America by Atlee in 1854, and again by Gaillard Thomas in 1870. It has been especially favoured by German operators, of whom Dührssen, A. Martin and Schauta operated through the anterior fornix, Mackenrodt, Bumm and Döderlein through the posterior. Döderlein out of 450 ovariotomies performed no less than 24% by the vaginal route. This method of operating, however, is only suited for simple, small or cystic tumours. In some cases it has been found necessary to complete the operation by the abdominal route, in others to remove the uterus. The frequent necessity of tapping the cyst has disadvantages, and the Pfannenstiel abdominal incision almost completely removes the risk of subsequent hernia and avoids an unsightly scar. A. Martin, who died on November 26, 1933, in his eighty-seventh year, out of 131 vaginal ovariotomies only lost two cases.

Remarks.—Since the centenary of ovariotomy a quarter of a century has passed, during which some technical improvements have occurred, such as the general adoption of the aseptic system; the more frequent operating on malignant, infected and complicated tumours, which sometimes require enterectomy or hysterectomy; the use of radiation in diagnosis and treatment; the removal of the tumour and leaving the rest of the ovary behind; and the insertion of grafts, or the injection of hormones when both ovaries have been removed. For ovarian tumours impacted in the pelvis during pregnancy, Cæsarean section is still too frequently employed. The long incision, used by McDowell, is generally used, as it avoids infection from the tumour or its contents. Covering the raw surface of the incision during the operation

prevents local implantation. The pedicle, unless securely tied, may give rise to secondary hæmorrhage; it is especially liable to occur when ovariotomy is done during pregnancy, in which case the vessels should be tied separately. In the only case of secondary hæmorrhage after ovariotomy which I have personally met with, the wound was opened and the pedicle retied. The patient, who had lost much blood, recovered and subsequently bore children. The pedicle may be a source of danger from adhesion of the intestine to it: I have lost a patient from that cause. Every care should be taken to cover raw surface with peritoneum. I have treated a few pedicles with the electric cautery-clamp, but have abandoned that method owing to the length of the burnt scar. A few have been treated by isolated ligature of the vessels and closing the peritoneum over the ligated portions with a purse-string suture. The round ligament and Fallopian tube also have been used to cover the pedicle.

Infection by tetanus (of which Spencer Wells had three instances) and bursting of the wound have disappeared from modern ovariotomy with the use of sterile instruments and stitching the abdominal wall in layers with non-absorbable ligatures (silk, thread, silkworm-gut). In addition, "stay-sutures" of silkworm-gut, tied over gauze, are used to obliterate dead spaces and prevent the formation of hæmatoma in the wound and discharges, which are not uncommonly met with when catgut sutures are employed. Drainage, by means of a glass or rubber tube, was frequently employed by the earlier ovariotomists, but is now rarely required, and only for hæmorrhagic, malignant, or incomplete cases, for which iodoform-gauze may be used.

During the last twenty-five years a great deal of research has been carried out in studying and classifying ovarian tumours—adenoma, adenofibroma, fibroma, myoma, actinomycosis, osteoma, chondroma, endometrioma, sarcoma, carcinoma, chorionepithelioma, teratoma (simple and malignant), papilloma, pseudomyxoma, "struma," neuro-epithelioma, granulosa-cell, endothelioma and luteoma. The pathogenesis and the malignancy of some of these tumours are still a fertile field for research.

If we look backwards for a century and a quarter some valuable lessons may be drawn from a study of the gradual development of ovariotomy. Much courage was required in the patient in pre-anæsthetic days to enable her to submit to an operation previously thought to be impossible or uniformly fatal, and in the operator who had no previous experience to guide him. When he succeeded he was often disbelieved and when he failed he was vituperated, not only by individual rivals, but also by professors and academies of medicine. It is interesting to note that it was not in the big cities but in the free air of the country that the first bold experiments were made, and mainly in countries where men were free to think, speak, and act, and not prone *jurare in verba magistri*. Of the "big five" nations, it was in America and England that the pioneer and chief promoters of the operation were found. In France and Italy only two ovariotomies had been performed up to 1860. In Germany, in 1843, Dieffenbach spoke of the operation as "sheer murder," and in 1854 Professor Scanzoni of Würzburg, having reluctantly operated on a case which terminated fatally, declined to operate again until 1869, and it was only after the adoption of the Listerian system by Schroeder, and German surgeons had come over to study the operation in England, that ovariotomy became popular in Germany.

The long incision used by McDowell, the short incision suggested by W. Hunter, dispensing with wound sutures, the use of the cautery and clamp, fixing the pedicle in the wound (Stilling in 1848, Duffin in 1850), long ligatures brought out of the wound, short ligatures (of leather, catgut, silk, or metal) dropped into the peritoneum, flushing, drainage—all these had been tried and their lessons learned. Still "the peritonitis beat them." It was only after the adoption of Listerism that this danger was eliminated and ovariotomy established as a safe operation, leading to a wide extension of abdominal surgery.

For the study of the early history of ovariotomy the most valuable records are those published by the American Gynecological Society in 1909, which I have utilized freely in this communication, and in Spencer Wells's "Ovarian and Uterine Tumours" (1882).

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Section of Epidemiology and State Medicine

President—J. D. ROLLESTON, M.D.

[*March* 23, 1934]

Problems in Hospital Administration arising out of the Local Government Act, 1929

By W. ALLEN DALEY, M.D., M.R.C.P.

IT is now nearly four years since the large local authorities of this country, namely, the county and county borough councils, became responsible for the public hospital provision of their areas, and sufficient time has elapsed to make it profitable to review the position, to consider our successes, and, perhaps even more profitably, our failures, to examine the directions in which we are travelling and endeavour to assess the progress made.

It is desirable in the first place to set out briefly the reasons, on the medical side, which led to the enactment of the Local Government Act.

The Guardians of the Unions which had been formed in 1834 had a statutory responsibility for the relief of the destitute. Many of the destitute were ill and were provided with domiciliary treatment by a district medical officer. Others, though not actually destitute in the strict financial sense of the word, were unable to obtain at home the medical treatment which they needed and were, therefore, technically "destitute," and these, together with those of the actually destitute who required in-patient treatment, were admitted to the sick wards of the institution which was, at that time, called "the workhouse."

For Poor Law purposes the country was divided into Unions, and these again into Parishes, but a movement was afoot to divide it in a different way for the purposes of sanitary and public health administration. Charters of incorporation as municipal boroughs were given to various towns and by 1872 the whole country was divided into sanitary districts, i.e. boroughs, urban, and rural districts. In 1889 county and county borough councils were formed. From 1889, therefore, we have had county boroughs with responsibility for administering in their areas the Public Health and other special Acts relating to public health, and outside the county boroughs, there were the administrative counties, where the duties and responsibilities under the Public Health and similar Acts were apportioned between the county councils and the sanitary authorities. In 1899 the metropolitan boroughs were formed and became the sanitary authorities for their districts.

All the large public health authorities took their functions seriously. Their

first inroad into the work of relieving medical destitution, up to then done by the Guardians, was to provide fever hospitals. This movement started some fifty or sixty years ago. It was argued that as the removal of infectious cases to hospital was primarily to prevent the spread of infection in the community it was a public health and not a Poor Law function, and that the "stigma" of pauperism should not be imposed. It should, however, be added that in London the fever hospitals were provided by a Poor Law authority, the Metropolitan Asylums Board, and it was specifically declared that admission to one of the Board's fever hospitals was not Poor Law relief. Under the Public Health Act, 1875, local sanitary authorities were empowered to provide general hospitals, but this power was only taken advantage of by the towns of Widnes and Barry, where an accident hospital was administered by the borough council, until, just after the Great War, the Bradford City Council took over a former Poor Law hospital in that city which, during the war, had been used for military purposes, and administered it as the first large general municipal hospital in this country. Since the beginning of this century, developments have occurred in many other directions, with the result that public health administrators realized that the old régime was coming to an end. Local health authorities became increasingly concerned with the provision of medical attention, in various aspects, for the inhabitants of their areas. By this time, most of their work in eliminating the grosser evils of insanitary environment, particularly in urban areas, had been completed, and they began, as the next step, to devote themselves to the care of the health of the individual. In the school medical service they established clinics for minor ailments, for the prescription of glasses, for aural cases, dental work, and for orthopædic and rheumatic cases, and provided beds for the operative treatment of adenoids and enlarged tonsils. Some authorities established "hospital schools" for orthopædic and rheumatic cases. Under the National Tuberculosis Scheme, sanatoria and hospitals for the tuberculous, in addition to tuberculosis dispensaries, were established; under the Maternity and Child Welfare Act, infant welfare clinics, ante-natal clinics and maternity homes and hospitals. Under the Venereal Diseases Scheme, clinics and in-patient treatment which, at first, were in nearly every area at a voluntary hospital, were established, but many are now provided in *ad hoc* premises and quite independent of voluntary hospitals. Again, the authority under the Mental Deficiency Acts established colonies and institutions. But, in all the instances which I have given of the provision by health authorities of medical treatment, the Guardians, under the statutory necessity of providing treatment for those medically destitute, continued at the same time to deal with a certain proportion of precisely the same ailments and classes of patient, and, with the development of medical science, many of the more progressive Boards of Guardians built or provided, by adaptation of other buildings, modern hospitals which were administered, under the Poor Law Acts, as hospitals under a medical superintendent, and not as workhouses or "mixed institutions" under a "master." Thus, although the health authorities at this stage had taken over from the Guardians cases of smallpox, scarlet fever and diphtheria, and the "isolation" blocks of the workhouses were relieved to this extent, cases of measles and whooping-cough were still being treated by the Guardians. Furthermore, the Guardians still remained the vaccination authority, despite the fact that the sanitary authority was responsible for dealing with an outbreak of smallpox in its area. The Guardians also provided wards for the tuberculous and the venereal, and for maternity cases, and some even retained mentally defective persons under their own care. These overlapping functions were administratively bad, and ever since the date of issue of the Reports of the Royal Commission on the Poor Law in 1909, public attention was focused on the matter.

The MacLean Report in 1918 summarized the whole position very clearly, but it was not until 1929 that Parliament finally agreed to take steps with a view to the "break-up" of the old Poor Law system, abolished the Guardians and transferred their functions to the large public health authorities.

An important object of this legislation was to dovetail into one administrative scheme the medical work of the Boards of Guardians and that of the public health authorities. This has turned out to be by no means an easy matter. The difficulties met with, however, vary enormously in different areas, and it will undoubtedly be some years yet before the dovetailing process can be regarded as reasonably complete, even in the best administered areas. In the first place, the number of hospitals or institutions transferred to individual health authorities varied from none, in towns where the Poor Law institution formerly served two or more towns or a town and a large county area, to 74 in the case of the County of London. Again, the nature of the accommodation for the sick varied from a few small and ancient wards in a mixed institution to fully equipped modern hospitals.

The Local Government Act makes provision for an authority to "declare" that some particular service may, for the inhabitants of that area, be provided entirely under the Public Health or other special Acts, and not under the Poor Law Act. This has the effect of removing entirely from the purview of the Poor Law system those who are covered by the terms of the declaration. For example, an authority may declare that all blind persons shall be dealt with under the Blind Persons Act, and if such a declaration is made, no blind person would receive indoor or outdoor *relief*, but would be referred, if application were made to a relieving officer, to the Welfare of the Blind authority, who would arrange for admission to a home for the blind, grant an allowance under the scheme made under the Blind Persons Act, or render assistance in some other form. Such a declaration involves the making of arrangements, apart from the system of outdoor medical relief, for blind persons who need domiciliary medical attention from a public authority. If a declaration is made with regard to the tuberculous, no patient requiring in-patient treatment primarily for tuberculosis can be retained in a Poor Law hospital. Tuberculous patients can, of course, even if a declaration is made, receive outdoor relief, because the special Tuberculosis Acts do not authorize a public health authority to make financial grants for maintenance to the tuberculous. The question of the supply of extra nourishment might give rise to difficulty.

It will be quite clear from the above that the making of a declaration is not to be undertaken lightly. The authority proposing to make it, and the Ministry of Health, must be perfectly satisfied that there are adequate facilities for dealing with every person in the area for whose benefit the special Act is intended. To take, as an instance, the case of a declaration concerning tuberculosis, under the Tuberculosis Act: the authority has discretion as to which patients shall be granted in-patient treatment in a hospital or sanatorium, and, if a patient under treatment in a sanatorium is obstreperous or a disturbing influence on other patients, they can discharge him. If no declaration has been made, the man, if still in need of in-patient treatment, applies to the relieving officer who arranges for his admission to the Poor Law hospital. If, however, a declaration has been made, if there is no "appropriated" hospital to which he can be transferred and if the authority has only one hospital for the tuberculous, such a man would probably have to be taken back, if his medical condition required it, into the same hospital from which he had been recently dismissed. I need not labour the point by taking other instances of difficulty, but, particularly in the smaller counties and county boroughs, it would

appear that formal "declarations" are better delayed until the full implications have been thoroughly investigated.

But, so far as ordinary hospital treatment is concerned, a step can be taken which removes the hospitals from administration under the Poor Law and yet does not involve the making of a "Declaration" that all sick people will receive hospital treatment under the Public Health and not under the Poor Law Acts. This is called the "appropriation" of a hospital. This curious word is used because it is necessary to invoke a section of an Act of Parliament which authorizes a local authority to "appropriate" land or a building for some purpose other than that for which it was acquired. Thus, a local authority can "appropriate" for use as a hospital administered under the Public Health Acts a hospital or other building which is being used as a Poor Law hospital or institution.

There are advantages and disadvantages in "appropriation," and these have been hotly debated since 1930 in many Council chambers.

From the point of view of the patient, the great advantage of "appropriation" is that he can be admitted simply by application to the medical superintendent, which is usually made for him on the telephone by his doctor. He has no need to apply to the relieving officer or to secure a relieving officer's order before his admission to a hospital can be obtained. I must, however, not omit to mention that, under the Public Assistance Order, a medical superintendent of a Poor Law hospital can admit a patient to the hospital, in emergency, on his own responsibility, but the point to be emphasized is that the *normal* method of seeking admission to a Poor Law hospital is through the relieving officer, and after "appropriation" it is through the medical superintendent; the relieving officer no longer has any concern in the matter unless, of course, a destitute person applies to him for medical attention, when it is his statutory duty to see that he is provided with it. The relieving officer can do so by securing the admission of the patient to an "appropriated" hospital. A further point is with regard to recovery of the cost of treatment. In a Poor Law hospital the cost of treatment and maintenance is "indoor relief," and the amount the "liable relatives" are required to pay is dealt with under the Poor Law machinery. In an "appropriated" hospital the patients (or those legally responsible for their maintenance) are required to pay the cost of treatment, or are assessed to pay what is considered reasonable having regard to their financial circumstances. Though this work of assessment may be conveniently done in a Public Assistance Department, efforts are, I know, being made in some areas to separate this assessment work from that for the provision of relief, and to obtain the necessary information by means of a system of hospital almoners. When a hospital is "appropriated" the administration is freed from the hampering restrictions of the "Public Assistance Order." The authority can make its own rules for the management of the hospital and has full control over the staff.

In effect, however, "appropriation" of a hospital and its administration under the Public Health Acts, means the provision of yet another medical service, namely, hospital treatment, which can be provided under two separate Acts, namely, for all inhabitants of the district under the Public Health Act, and for those destitute of medical aid under the Poor Law Act.

There are, however, points on the reverse side. The most important is with regard to the recovery of the cost of treatment of extra-district patients. Safeguards are provided in the Poor Law Act to prevent the Public Assistance Rate of one area being charged with the cost of relief provided for the inhabitants of another. There is the Law of Settlement, and, in cases of accident or sudden illness, Section 80 of the Poor Law Act, 1930, enables the charge to be recovered from the authority deemed by the Act to be responsible for its payment. In the case of "appropriated"

hospitals the legal position is by no means clear, and further legislation or a High Court decision will probably be found to be necessary. There is, also, the point that certain action under the Mental Deficiency Act, the Vagrancy Act, the Bastardy Acts, and with regard to the adoption of a child by the Council, can only be put into operation when the person concerned is in receipt of Poor Law relief, and these powers cannot, therefore, be applied in the case of a "municipal" patient in an "appropriated" hospital.

"Appropriation" of a separate Poor Law hospital is administratively easy but certain authorities are endeavouring to "appropriate" the hospital section of a mixed institution and administer it as a separate hospital under the Public Health Acts. Though the effort is praiseworthy, the presence of a single kitchen, laundry and one set of administrative offices makes the effective separation which is required by the Act difficult.

Severance from the Poor Law system by "appropriation" of some, at least, of the hospital accommodation transferred to the new authorities is, however, essential if the satisfactory welding together of the various medical functions of a Public Health authority is to be accomplished. Examples of what has been done already may be given. A school medical department with a long waiting list of children in need of operative treatment for adenoids and enlarged tonsils has admitted patients for this treatment to "appropriated" hospitals, particularly in the summer months when the pressure on the municipal hospital beds is relaxed. Tuberculous patients, or those suspected to be tuberculous, can be sent into an "appropriated" hospital for observation and classification. Again, patients who have ceased to improve in a sanatorium or special tuberculosis hospital can be transferred to the tuberculosis ward of an "appropriated" general hospital. Formerly, such patients often refused transfer when they knew it was to a Poor Law hospital, but if the general hospitals are "appropriated" the tuberculosis or other officer can control all the beds for tuberculosis in the area, and transfers are thus facilitated. Another benefit already seen is in connexion with the provision of maternity beds.

Many Maternity and Child Welfare authorities built small maternity homes or hospitals, or, more commonly, adapted large private houses for such use. As these are comparatively small, the cost per patient per week, owing to overhead and other charges, is high, and they lack the presence on the spot of medical and surgical consultant opinion and often even of a resident medical officer, and a pathological laboratory, all of which are obtainable in a municipal general hospital.

All county boroughs and most county councils administer the Maternity and Child Welfare Act. They make increasing use of the maternity wards in "appropriated" general hospitals, and ante-natal and post-natal clinics are being arranged in connexion therewith; that is to say the maternity and child welfare work is now more and more being linked up with the general hospital service. Even in areas like London, where the county council is not the Maternity and Child Welfare authority, coördination of the work of the two authorities, namely the London County Council as the Hospital Authority and the Metropolitan Boroughs as the Maternity and Child Welfare authorities, is being effected. In Edinburgh the work of the V.D. units in all the municipal hospitals has been coördinated with that of the V.D. treatment centres, and placed under the same medical direction.

A further consolidation of the Public Health services has taken place in some areas by the inauguration of a system by which particulars of all school children treated in the municipal hospitals are forwarded to the school doctor, who arranges

appropriate reinspections at school. Many school medical departments have recently established special clinics for rheumatism. Many of the children require hospital treatment in order to prevent cardiac involvement or to limit the extent of such involvement. The provision of combined clinics for child welfare, school medical work and tuberculosis dispensaries in connexion with the out-patient departments of municipal hospitals is also under consideration in certain areas.

The above are some of the ways in which the new hospital services can be linked up with the pre-1930 Public Health services. But the administration of a municipal general hospital service, though it helps to solve some of the old problems, brings many new ones in its train.

The first and fundamental one is how much hospital accommodation is needed for an area, and what should be its nature?

Inquiry shows that the numbers of beds per 1,000 of the population vary enormously from country to country and from town to town, e.g. including mental hospitals, the total beds provided per 1,000 of the population in England and Wales in 1929 was 9·47. (In London it is 19·57, but this includes 3·37 in voluntary hospitals, one-third of whose patients come from outside the County.) In Scotland the figure is 7·86; in U.S.A. 7·45; in Germany 8·05; Holland 6·5; Poland 2·37. These figures, except for London, are taken from a paper by Dr. Mansholt, Holland.[1]

It appears that, up to a point which cannot be determined with any accuracy, the demand for admission to hospital tends to grow with the provision of satisfactory accommodation of good local repute. Such factors as housing accommodation, overcrowding, the incidence of endemic or local industrial diseases, and the number of motor accidents which occur in the vicinity, are to be borne in mind when determining the hospital needs of an area.

With regard to the nature of the accommodation required, the extent to which specialization is possible or desirable depends on the size and nature of the area. In London and the large cities and urbanized portions of county areas, much can be done to provide special hospitals and special units in general hospitals which would be quite impossible in small or middle-sized towns or in rural counties. Examples of special hospitals, apart from fever hospitals and sanatoria which are provided everywhere, are:—children's country hospitals for cases requiring prolonged treatment ("hospital schools"); hospitals for chronic skin diseases; hospitals for chronic eye diseases; hospitals for ophthalmia neonatorum and congenital syphilis; epileptic colonies; convalescent homes.

Special units are required in only one of a series of hospitals for conditions where special skill is needed on the medical or surgical side, or where the equipment required is very expensive. Examples are: plastic surgery, chronic rheumatism, diseases of the thyroid gland, congenital deformities, deep X-ray and radium therapy, puerperal fever, and thoracic surgery. Another reason for a special unit is the need for collecting as much clinical material as possible for purposes of investigation and research.

There are other special units which are required, not in every hospital but for each group of hospitals, such as maternity wards, V.D. wards, tuberculosis wards, and a mental observation unit.

It is, however, quite impossible to generalize on the nature and amount of municipal hospital accommodation required for an area. There can be no uniformity, for many reasons. One of great importance is the number of beds in voluntary hospitals, and consultation with representatives of the voluntary hospitals is required by the Local Government Act when any extension of municipal hospital facilities is

1 Read at First International Hospital Congress, Atlantic City, June 1929. [Published by the New York Academy of Medicine.]

contemplated. On the other hand, voluntary hospitals should consult with the local authorities of their area when they propose to provide more beds or new departments.

As the result of the formation in London, under the Act, of a committee representative of the voluntary hospitals, a detailed survey has been made of the voluntary hospital provision in London and the use made of it. The result of the survey has been published, as has also that of a complementary survey of the municipal hospital accommodation. These are most valuable documents and similar information for each separate area of the country would be useful.

On the question of the number of beds, there was a general impression some years ago that, while voluntary hospitals had enormous waiting lists, the Poor Law hospitals had large numbers of empty beds. The former is undoubtedly true, but the position in the Poor Law hospitals, which has been much exaggerated requires a little explanation. A voluntary hospital can decline to accept any patient and does so, naturally, when its beds are full. On the other hand, a Poor Law hospital must admit "on demand" those who are medically destitute, including those sent on from a voluntary hospital. Incidentally, this obligation is not removed by "appropriation" of the hospitals. There are high and low tides of disease, and the municipal hospitals must be prepared for the high tide. In practice, when the high tide comes, as it does in January, February and March each year, or when influenza is prevalent, the municipal hospitals are sorely pressed for room, and it is only when the tide is low, as in the summer months, that there is surplus accommodation. At that time, staff are on summer leave and painting and repairs are undertaken. "Surplus" accommodation is, in most areas, non-existent for the greater part of the year, though there are periods when more patients could be accommodated. In certain towns, particularly where the honorary staff of the voluntary hospital are attached to the municipal hospital, the municipal hospitals have, at these periods of low demand, dealt with patients on the waiting list of the voluntary hospital.

Another problem to be settled is the extent to which it is desirable, or possible, to relieve the pressure on accommodation in acute hospitals by providing convalescent or recovery hospitals in the country or at the seaside, to which those recovering from acute illnesses can be transferred for the completion of their treatment. Another point bearing on bed accommodation is the question of out-patient departments, and there everyone will agree that it would be a mistake for municipal hospitals to develop out-patient departments on the lines of the large voluntary hospitals with their hundreds of thousands of attendances every year. But out-patient departments have considerable value as diagnostic centres, for special treatment, and for continuation treatment after the conclusion of in-patient treatment. The following rule of the London County Council sets out the type of case which may be dealt with in their out-patient departments:—

(1) Casualties, accidents and other emergencies.

(2) Continuation or after-care treatment of former in-patients requiring further treatment after discharge.

(3) Consultation concerning patients referred by any duly authorized medical officer of the Council.

(4) Ante-natal and post-natal examination and treatment.

(5) Patients entitled to out-door medical relief.

(6) Special examination and/or treatment of cases or classes of cases authorized by the medical officer of health.

The provision of facilities for home nursing can also effect some reduction in the number of beds required by enabling certain patients, who would otherwise have to

be admitted to hospital, to remain at home and others to be discharged from hospital at an earlier date.

Much discussion has occurred with regard to the care and treatment of the chronic sick. Some hold that they are best looked after in a Poor Law "mixed institution," others would go so far as to exclude all chronic sick from the general hospitals which, they consider, should be reserved for the acute sick, and they would provide separate hospitals for the chronic sick, while yet others think that there are distinct advantages in treating a certain proportion of chronic sick in general hospitals. It certainly does not appear to be consistent with the policy of breaking up the Poor Law that large authorities should retain mixed institutions with their range of inmates from able-bodied, through the infirm, to the chronic sick, and in some sick wards, even the acute sick. We must concede, however, that in rural areas this may be inevitable. A decision as to whether the admission to, or the exclusion from, general hospitals of the chronic sick is the better policy cannot easily be reached. The main argument in favour of separate hospitals for the chronic sick is that the cost of maintenance per patient-week is less, mainly because of the lesser requirements both as regards staff and the type of accommodation and equipment necessary, and it may be argued that an expensive bed in an acute hospital is used extravagantly when occupied by a "chronic sick" patient. In such a discussion, definition of terms becomes advisable though not easy, and the acute sick may be regarded as those who will improve sufficiently to be able to be discharged within a reasonable period, or who are suffering from some condition which will probably lead to an early death.

"Reasonable" and "early" are, of course, indefinite terms but may, for practicable purposes, be regarded as within from one to two months. Chronic cases, on the other hand, are those who are likely to require continuous medical and nursing care in a hospital or institution for a prolonged period, i.e. for some months or even years.

With regard to staffing, on the first admission of a case which ultimately turns out to be chronic, it is desirable that a full clinical investigation should be made. This is a point in favour of their first admission to a general hospital with a full medical staff and facilities for investigation, including a laboratory. The amount of nursing required for the chronic sick depends, of course, entirely on the nature of the conditions under treatment. In the case of the incontinent it may approach that required in a ward for those acutely ill, but, speaking generally, the nursing need not be of the quality or quantity requisite for an "acute" ward. There is, however, a point on the nursing side which is worthy of mention. Most municipal general hospitals are now also training-schools for nurses. When nurses, after training, go out into the world they often nurse "chronic" cases. It is desirable, therefore, that during the course of their training, they should have had experience of all types of work, including the nursing of the chronic sick, and this is another point in favour of admitting a certain proportion of such cases to a general hospital. Again, it has been found more difficult to recruit medical and nursing staff for a hospital devoted entirely to the chronic sick than for an acute hospital, as the work is said to be not so interesting. If a special hospital for chronic cases is provided, every effort will be needed to keep it as bright and cheerful as possible. On the structural side, the special departments need not be of so elaborate a nature as are required in an acute hospital.

Other matters arising out of the change of administrative control of establishments provided under the Poor Law Act may now be mentioned briefly.

In many areas it was found that there was no officially defined relationship between the district medical officer—who, amongst his other duties, issued, through the relieving officer, admission orders to hospital—and the medical superintendent. It is desirable that a closer coöperation should exist between these two officers and that the district medical officer should work in future under the general supervision of the medical superintendent. It would require a paper all to itself to discuss the much-vexed question of how district medical work can best be done, e.g. whether by whole-time staff, either engaged entirely in district work or employed also as assistant resident medical staff at a hospital, by part-time specially appointed general practitioners, or by a system which embraces every practitioner in the area who is willing to do the work. All these systems have their advocates, except, possibly, that of the whole-timer doing district medical work only.

A considerable number of residential Poor Law schools were transferred to the county and county borough councils in April 1930, and they brought with them their own problems.

The best method of controlling infection in them is by no means finally settled. The former method of passing all new entrants through a quarantine block often failed, and it required much staff and accommodation. Methods involving daily skilled inspection of all new entrants until the maximum incubation period has passed are being tried. It cannot be said that so far its success is more than "partial," though as a result of further experience on the part of all concerned the initial difficulties may eventually be overcome.

But, in these schools, immunizat on methods against diphtheria and scarlet fever have been used with remarkable success.

Many of the schools were provided with fairly large hospital blocks, and quite serious illnesses were treated and operative procedures undertaken. In my opinion, if a general hospital is within easy access, it is advisable to transfer the acutely ill child to a hospital where constant medical attention and skilled nursing are available.

I should now like to speak of the transferred hospital service as a field for investigation and research. Even in the smallest county or county borough, the whole public medical service of the area is now under the same control, cases can be followed through and clinical research undertaken. In the largest authorities the opportunities for investigation afford much wider scope.

In the first place, there is much investigation to be done in the question of hospital buildings. Have we yet designed the ideal ward block? What are the proper amounts of wall space and floor space for patients of different types? There are literally hundreds of points in the design and structure of a hospital on which there is no authoritative opinion. A voluntary hospital or a small local authority may have to pay dearly for their inexperience, but a large authority controlling many hospitals can afford to investigate fully and to experiment for the benefit of themselves and others.

Then there is standardization of equipment and mass purchasing. In the London area before April 1930, there were 26 standards and 26 sets of contracts for the various hospitals. By careful study and trial, standard samples of many items of hospital equipment can be agreed with all concerned, and the resultant reduction in cost, without in any way sacrificing efficiency, has been considerable, amounting in the case of one authority to 20%.

Next there is the study of hospital administration, and for this purpose detailed costing returns should be prepared for each hospital.

Most illuminating sidelights on administrative problems are revealed by these returns and the resulting investigations. To take such simple things as staff dietaries, it by no means follows that where the cost is highest the staff is the best fed. Returns showing the amount of waste food or of breakages are all worthy of study.

A word of warning is, however, necessary with regard to the misuse of costing tables. No two hospitals are exactly alike, and costing returns must be considered in relationship to all the varying factors involved. They give pointers which indicate the need for inquiry, but not necessarily for action to cut the item down.

In view of the fact that the cost of staff represents approximately 50% of the total cost of maintaining a municipal hospital, exhaustive study of ratios of staff of various grades for different types of hospital is essential. Also problems of clinical staffing need consideration, such as the exact rôle of the consultant, the whole-time specialist, the house physician, house surgeon and clinical assistant. Another question is the training of future medical superintendents.

In a very large hospital the medical superintendent may be almost entirely engaged in administrative work. But, if he is provided with adequate lay assistance, he can still take part in some clinical work. In a large hospital service, the senior resident staff, though mostly engaged in clinical work, have opportunities of learning administration, and so preparing themselves for appointment as medical superintendents. It is, however, wasteful to train a man to be an expert clinician or operating surgeon, and then employ him almost entirely in administrative work. There is a tendency for this to happen in areas where the administrative posts are remunerated much more highly than the clinical, and the appointment of whole-time specialists at salaries which compare with those of administrators is well worth consideration.

An instance of a useful piece of coördination made possible as a result of the Local Government Act is the amalgamation in London, under the control of the County Medical Officer, of the accident ambulance service, formerly administered by the Fire Brigade, and the ambulance services of the Metropolitan Asylums Board and the various Boards of Guardians.

Facilities for pathological investigation in a large hospital service are essential, and the London County Council has provided five group laboratories and a central histological laboratory, together with numerous hospital laboratories, in addition to the two group laboratories and an anti-toxin establishment which it inherited from the Metropolitan Asylums Board. All these have been coördinated into one pathological unit.

The facilities for teaching clinical medicine, ect., at municipal hospitals should also be mentioned. Each medical school in London is now linked up with one or more municipal general hospitals. The students visit the hospitals regularly, and are instructed by a member of the staff of their own school. Clinical material in obstetrics for the teaching of medical students is in many schools inadequate, and the maternity wards of certain municipal hospitals are being used to supply the deficiency.

Classes of post-graduates are also held, and there are clinical demonstrations attended by general practitioners. The scheme for the use of the London County Council's Hammersmith Hospital as the post-graduate school of the University of London is well known to all interested in medical education.

The provision made for clinical research at various units has already been mentioned, but it may be added that statistical research on a large scale can now be undertaken.

For each of the 200,000 patients admitted to the London County Council hospitals in the course of a year, the essential features of the case are set out on a "Powers Samas" card. These can be investigated by mechanical tabulation and the relationship of various diseases to age, occupation, result of treatment, etc., worked out. Further, by this centralized system, if some rare conditions are to be investigated, the cards of those suffering from the complaint over a series of years can be sorted out and the actual case papers obtained from the hospitals. Special cards can be used for maternity, tuberculous, fever, etc., cases.

It must not, however, be thought that this study of costs and administrative procedures will lead to a sterile uniformity of practice. Every hospital has its own individuality and its own problems which must be solved locally, but in a large hospital service there are many things in which uniformity is desirable, e.g. standard dietary scales of *quantities*, the local initiative being occupied in devising their own menus; again, there is no reason why bandages and surgical dressings should not conform to certain standards of quality depending on the nature of the use to which they are to be put. But this standardization need not stultify progress; in fact, in hospital administration, there can be no "stasis." A wide-awake and efficient central administration welcomes suggestions from the periphery, and circulates particulars of the good ones so that they may be put into operation elsewhere. In fact, in one area, where the hospitals were recently circulated and suggestions for economy asked for, literally hundreds were sent in and many valuable recommendations resulted. Lastly let us look at the effect of the Local Government Act on the progress of preventive medicine.

Curative medicine is often regarded as a more attractive study than that of preventive medicine. The results of the former are often sudden, dramatic and eminently satisfactory. The patient at death's door with pneumonia or appendicitis leaves the hospital a few weeks later practically restored to health. The triumphs of preventive medicine are unknown to all save the few who have studied medical history. The arrested epidemic does not stir public opinion. Little notice is taken of a gradual reduction in the incidence of, or death-rate from, any particular disease. There is a danger in the present situation that we too may become so immersed in the fascinating and absorbing problems of administering hospitals and curing disease that we may forget that our primary function is to preserve health and prevent disease. As a corrective, however, of this attitude of mind, the cost of hospital treatment does bring home to us, and to the members of Local Health authorities, the enormous burden on the community caused by ill-health. Once the immediate difficulties incidental to the transfer of such a complicated piece of administration have been overcome, then it will be our bounden duty to endeavour to ascertain what are the preventable conditions which are filling our hospitals, and what steps can be taken to combat them. The stage when these investigations will commence need not be much longer delayed. In fact, in different parts of the country, they are probably going on now. When some tangible results of the investigation of these great problems of preventable disease appear, then, indeed, will the work of the framers of the Local Government Act be fully justified. In the meantime, we can be quite content; we are in the process of moulding a large additional service on to the general public health service of the community—in some areas the addition is much larger than the original.

The change-over took place so easily that the majority of the public, and even of the patients, were unaware that on April 1, 1930, there had been a change of administration. The new administration has, however, brought the public hospital service into the limelight of municipal and medical politics. The voluntary hospitals show a new interest in the municipal hospitals. There is closer coöperation in every way. As already mentioned, the municipal hospitals are used for supplementing the

teaching of medical students by linking up with the great teaching hospitals. Hospital administration as a science is receiving more attention. To revert to the title of this paper, several problems have been referred to, few, however, are solved. Certain directions of thought and progress have been mentioned. The motto has been "hasten slowly," but much solid work has been done ; staffing on the medical and nursing sides has been greatly improved, unsatisfactory wards have been modernized, new hospitals have been acquired, existing hospitals extended, facilities for special treatment and for pathological examinations greatly increased and improved, and classification of hospitals and units has been effected. All this is satisfactory but much still remains to be done. As mentioned before, however, perhaps the greatest tribute to the esteem in which the public hospital service is held is that the new Post-Graduate School, incorporated by Royal Charter and approved by the University of London, is to be located at a municipal hospital in Hammersmith. With the final thought of all that that means to the public hospital service I will finish.

Section of Dermatology

President—HENRY MACCORMAC, C.B.E., M.D.

[*May* 17, 1934, *continued*]

Poikilodermia Reticularis Atrophicans (Jacobi).—W. N. GOLDSMITH, M.D.

C. F., a man aged 43.

Present condition.—There is a widespread, symmetrical, sheet-like alteration of of the skin of the trunk, thighs, axillary folds and arms. It is most marked on the abdomen and the inner aspect of the thighs. The patches, which are of irregular shape and mostly confluent, have a brownish-pink colour. There is very slight scaling. On the back there is a fine reticular arrangement, the meshes being white and the borders brownish-red. On the thighs the skin appears to be superficially crinkled into fine ridges and furrows: but when it is stretched it shows the same reticulate pattern as on the trunk.

History.—Duration eighteen months. The condition is now stationary. It began in the groins and on the abdomen and has always been symmetrical. A biopsy was made from the right thigh but, as usual in this group of disorders, showed only slight changes, apart from thinning of the epidermis, and nothing of diagnostic importance.

DR. J. M. H. MACLEOD said he did not consider this to be a case of poikilodermia atrophicans vascularis, but thought that it was parapsoriasis of the "en plaque" type. What Dr. Goldsmith had referred to as reticulation in parapsoriasis was familiar in cases of the variety known as parakeratosis variegata.

Lupus Miliaris Faciei.—W. N. GOLDSMITH, M.D.

J. R., a man aged 28.

Present condition.—There is a dense eruption of small, round lesions, confined to the face and grouped especially at the root of the nose, on the eyelid, in the naso-labial folds and on the upper lip and chin. They are brownish-red, slightly translucent papules, mostly not exceeding about 3 mm. in diameter. Many are capped by a small central crust or a little yellow spot looking like a pustule, but at no stage has pus been evacuated. There are also a number of slightly depressed pink scars, representing the sites of healed lesions.

History.—The eruption began on the right side of the upper lip six months ago, It extended across the lip and on to the chin. A month later the lower lids became swollen and similar papules appeared on them. The upper lids and the forehead became involved two months after the onset. The patient has had no previous skin trouble or other illness. There is no family history of tuberculosis.

Investigations.—General physical examination revealed no abnormality. Skiagram of chest normal. Blood Wassermann reaction, negative. Tuberculin reactions, intradermal with old tuberculin up to a concentration of 1 : 1,000, quite negative.

Biopsy, April 17, 1934.—A microscopical section reveals a characteristic tuberculous granuloma tending towards the sarcoid type of reaction, namely a preponderance of epithelioid cells and scantiness or absence of lymphocytes and giant cells.

Course of treatment.—There has been marked improvement during the past month under treatment by daily general carbon arc-light baths (including the face), cod-liver oil and malt, compound syrup of hypophosphites, and salt-poor diet.

Comment.—This is the condition first described by Tilbury Fox in 1878 as lupus follicularis. The chief clinical difference from lupus vulgaris is that each single lesion shows no tendency to increase in size. In this case the negative tuberculin reaction and the histological picture, with its absence of necrosis and giant cells, bring it into close relationship with sarcoids. The tuberculin reaction in most of these cases is weak or negative, but the histological structure in some cases is more classically tuberculous, and in other cases again (as in most of those diagnosed as acne agminata) there may be conspicuous necrosis and a non-specific dense cellular infiltration. The condition may be associated with erythema induratum-like lesions or papulo-necrotic tuberculides. Tubercle bacilli have only very rarely been found. Animal inoculation is generally negative but has been occasionally positive.

Lymphocytoma.—W. N. GOLDSMITH, M.D.

G.P., female, aged 46.

Present condition.—On the right cheek and the bridge of the nose are two rather ill-defined, raised, brownish-red swellings of somewhat rubbery consistency. On diascopy most of the colour disappears, but a few little grey flecks remain. The clinical appearance resembles that of sarcoid.

History.—Duration ten years.

Investigations.—In the expectation that the condition was sarcoid, a skiagram of the phalanges was taken, and the radiologist, Dr. R. W. A. Salmond, reported "a well-defined area of translucency at the base of the proximal phalanx of the second right toe as well as punched-out appearance at both internal cuneiforms." The tuberculin reaction, intradermal with old tuberculin, was strongly positive in a dilution of 1 in a million. This made sarcoid unlikely.

Biopsy.—Microsocpical examination showed beyond doubt the rare condition "lymphocytoma." There are sharply demarcated islands composed almost entirely of lymphocytes. Though the demarcation is so sharp, there is no trace of fibrous encapsulation and there is nothing suggestive of an inflammatory process. Dr. Freudenthal tells me that in some cases one can see appearances closely resembling the germ centres of lymph-glands, which suggests that these little tumours may represent abortive cutaneous lymphatic glands. The great sensitiveness to tuberculin and the clear space in the bone probably have no relationship with the lymphocytoma. I have only seen one other case. The patient had multiple tumours like these on her forehead. They disappeared completely under X-ray treatment. The blood was examined for leukæmia every six months for several years, but remained perfectly normal.

Bowen's Disease.—GODFREY BAMBER, M.D.

Miss B. H., aged 31, has had psoriasis intermittently since she was 7 years old. Ten years ago she noticed a pimple below the left breast. A larger area gradually became involved and became covered with scabs.

Present condition.—On the left side of the epigastrium there is a somewhat scaly oval area containing papules of various sizes, some of which were crusted.

Report on microscopical examination of a papule.—There is thickening of the horny layer with some parakeratosis. The epithelium shows a noticeable proliferation and down-growth.

Some of the cells show clumping of the nuclei and vacuolation. There is some œdema of the epidermis and the underlying corium with the formation of vesicles. The cutis shows lymphocytic infiltration. The histology is that of an epithelioma of the Bowen type.

Leprosy (Mixed Nodular and Anæsthetic).—F. A. E. SILCOCK, M.D.

E. T., a man, aged 48. Riveter in an engineering works. Was in the regular army for fifteen years; discharged in 1916 for wounds. Served in India for nine years of his time; last there in 1914, and has not been in any endemic area since.

Previous history.—Had "a touch of pleurisy on the right side" six years ago. Attack of herpes zoster on the left buttock four years ago; the scars of this are still visible. Otherwise general health has been good. There is the mark of an old shrapnel wound in the right groin, about two inches below, and internal to, the right anterior superior iliac crest.

Wassermann reaction, May 10, 1934, negative.

History of present disease.—No history of any skin rash, malaise, headaches, febrile attacks, &c. (other than those mentioned above) previous to the onset of shooting pains down the right forearm and in the region of the right elbow-joint. These pains he describes "as if someone had knocked him on the right funny bone." Two

Leprosy (mixed nodular and anæsthetic type).

years ago he noticed that the right little finger and the inner side of the right forearm were becoming numb. At the same time the nerve pains, already mentioned, became very severe in this region; they have since eased off. He frequently burns his right hand and forearm in the course of his work, without being aware of it.

He says that, in August 1932, a lump appeared on the left upper eyelid. In January 1933 this was excised in the hospital out-patient department. Following

this, three or four more similar lumps appeared on the left eyelid and cheek. These were excised at the same institution in August 1933. Afterwards several more nodules appeared on both sides of the neck, forehead, malar regions, chin, right hand and forearm, and a few (about five) on the left forearm.

Present condition.—General condition very good. Definitely marked "leonine facies." There are numerous diffuse nodules, varying in size from that of half a pea to that of a hazel-nut, scattered over the face and forehead, around both eyebrows, over the eyelids, both sides of the neck, the back of the right hand, along the right forearm, and some on the left forearm. There are a few along the nose externally. There are none on any of the mucous surfaces, as far as can be seen. Their consistency is firm: none have ulcerated yet, and the overlying skin varies in colour from normal to pink, red, and even purple at times. They are not tender to touch. Since treatment with sodium chaulmoograte and hydnocarpus oil was begun the tumours have shown a tendency to get larger at first. There is anæsthesia, both superficial and deep, along the inner side of the right forearm and both sides of the right little finger and hypothenar eminence, corresponding to the skin area supplied by the right ulnar nerve; I cannot make out any thickening along the course of this nerve.

There are also one or two small scattered erythematous patches on the body, but these are not marked at present.

This case illustrates the long latent period that may elapse before onset of the disease, there having been an interval of thirteen or fourteen years between the time when the patient left India and that at which the first symptom appeared.

Histological report on section of nodule from right forearm.—(Dr. I. Muende.) (1) *Stained hæmatoxylin and eosin.*—The epidermis is thinned and shows evidence of slight excess of pigmentation. The upper two-thirds of the corium is for the greater part replaced by large circumscribed zones of granulation tissue and strands of closely packed cells insinuated between the collagen bundles. This cellular tissue is composed chiefly of compact masses of œdematous, and at times slightly vacuolated, epithelioid cells and few lymphoid cells. The infiltration is well seen around the pilosebaceous follicles and sweat-glands.

(2) *Stained Ziehl-Neelsen method.*—Section shows presence of very numerous acid-fast bacilli, frequently arranged in closely packed bundles, and morphologically resembling Hansen's bacillus.

Smear from nodule shows presence of acid-fast bacilli; smear from nasal mucosa does not show any acid-fast bacilli.

The man's occupation is one in which he is liable to injure himself with hot metal, heavy machinery and tools. He has a wife and six children depending on him, and naturally he does not want to give up his employment. I am not compelled to notify the case to the authorities; if I did so, the patient would probably lose his job.

Dr. J. M. H. MacLeod said that he agreed with the diagnosis of leprosy. He regarded such cases as contagious, though requiring contact over a long period for the contagion to be effective.

Exfoliative Dermatitis.—L. Forman, M.D.

S. S., male, aged 35.

In 1926 had a generalized erythema of the face, arms and legs for six months, said to be seborrhœic dermatitis.

In 1931 he had another attack, involving the shoulders and back only.

In 1932 there was a recurrence involving the whole of the body. This cleared up in a few weeks.

In 1933 the patient had a relapse. The right antrum was found to contain muco-pus and was drained. The skin was much more moist after operation.

During the last ten months the patient has been in hospital. There was some improvement in March, but an almost immediate relapse followed, with a widespread mixed erythematous lichenoid and eczematous eruption, leaving numerous islands of normal skin on the face, chest and back.

The appearance of this eruption was very like a toxic heavy metal dermatitis, but there is no history of injection of bismuth or arsenic, and examination of the hair for arsenic was negative.

The skin is now exfoliating freely. The superficial glands are palpable and have increased slightly in size during the past three months.

Blood-count (27.2.34).—W.B.C. 15,000 per c.mm.; polys. 49%; eosinos. 18%; basos., 1%; lymphos. 21·5%; monos. 10%.

Blood-count (11.4.34).—W.B.C. 8,000; polys. 55%; eosinos. 13%; basos. 1%; lymphos 26%; large monos. 5%.

The lævulose tolerance test showed some liver deficiency.

Half an hour after 100 grm. of lævulose	0·135%
One " " " "	0·12%
One and a half hours after "	0·105%

Intradermal injections of Dick streptococcal toxin, of staphylococci, of the streptococcus from the patient's antrum, and of *Bacillus coli communis* gave negative results, but they were performed on a slightly erythematous skin. A microscopical section of the skin showed marked acanthosis, with œdema of the epidermis and corium, with some lymphocytic infiltration of the latter.

Pityriasis Lichenoides et Varioliformis Acuta.—J. E. M. WIGLEY, M.B., and GORDON HEGGS, M.D.

A healthy young man, aged 20, noticed, three weeks ago, an eruption on his face and trunk which itched considerably. He felt perfectly well at that time. A week later the eruption extended to the upper arms and thighs whilst clearing from the trunk. A week later it extended on to the forearms, legs and feet, whilst beginning to clear from the upper arms. With each spread of the eruption there was an exacerbation of the itching, but no malaise beyond a slight headache.

The patient has not been in contact with any variola or varicella cases and he has not himself had varicella. He shows the scars of effective vaccination performed ten years ago.

He now presents a polymorphic eruption, consisting of erythematous macules, reddish papules—some covered with a blood-crust and some with a brownish scale—and definite pustules. No definite scars are seen. The eruption is present on the forehead, eyelids, back of neck, "muzzle," area, limbs, backs of hands, and soles of feet. On the trunk and penis are seen disappearing lesions, consisting of slightly brownish macules, frequently covered with a brownish scale. There is slight general adenitis and there is moderate pityriasis of the scalp.

Temperature on admission, 100° F., pulse 110, blood-pressure 130/70. Physical examination completely negative. No congestion of throat. Spleen not palpable. Wassermann reaction, negative.

Blood-count.—R.B.C. 5,800,000; Hb. 105%; C.I. 0·9; W.B.C. 7,100; polys. 53·67%; eosinos. 2·34%; large hyals. 2·0%; lymphos. 42%.

The irritation accompanying the eruption and the affection of the soles of the feet are unusual features in this case. The appearance of the eruption in definite "waves," the generalized glandular enlargement, and the slight fever may be regarded as supporting the view of Buschke, Riehl, Pusey, Pollitzer, and others, that pityriasis lichenoides et varioliformis acuta is a separate and distinct entity. The probability of a specific virus as the cause is an attractive one.

Pathological report (Dr. Muende).—The first section was taken from a small pustule. There is a very large intradermal pustule, the roof of which is composed of a few layers of parakeratotic horn cells, except peripherally, where there is a thin horny fringe. The base consists of a very thin and compressed stratum malpighii, the cells of which exhibit a marked degree of intracellular, but comparatively little intercellular œdema, with considerable intercellular immigration of leucocytes, which are chiefly polymorphonuclear. Some of these epidermal cells show a marked eosinophilia, and resemble the balloon cells of herpes zoster; some others show large and occasionally duplicated nuclei with well-developed nucleoli. In the corium, the papillæ are very dilated and œdematous; the blood-vessels are engorged, and in the papillæ generally, and also around the hair follicles, there is a marked leucocytic infiltration, the cells here being chiefly of the small leucocyte type. The vesicle fluid contains a very large number of degenerated white blood-cells, together with fibrin.

In the second section, from a more advanced lesion, the cap of the pustule is now absent, and the stratum malpighii has completely disappeared in the central third of the base where there are now several large engorged blood-vessels. The corium beneath the pustule is comparatively free from cellular infiltration.

The third section is from a lichenoid lesion. Here the stratum malpighii has regained its normal appearance, but is now covered with a thick scale of alternating keratosis and parakeratosis. The papillæ are widely dilated and œdematous, and contain very large congested blood-vessels. There is also a sparse cellular infiltration in the papillæ and also around the superficial blood-vessels.

Lichen Amyloidosus.—A. M. H. GRAY, C.B.E., M.D.

The patient, a man aged 49, was first seen in August 1931. He presented on both legs, from the junction of the upper and middle thirds down to the hollows below the inner ankles, a number of raised, warty tumours, some isolated, others running together to form ridges or masses of irregular contour. The individual lesions, though varying in size up to that of a shilling, rose sharply from the surrounding—apparently normal—skin; they were dome-shaped and some had a central depression; they had a dull purplish colour and the surface was studded with large pits and covered with fine, dry scales. To the feel the lesions were very hard. The patient complained of considerable itching in them.

Biopsy was performed on 1.3.34, and Dr. Freudenthal reported as follows:—

"A piece of skin—4 cm. by ½ cm.—was taken from the left leg so as to include a prominent lesion, pitted in the centre. Corresponding to the clinical features the papillary body in the middle of the slide appears broader, the rete and the horny layer prominent. In the centre of this prominence is to be seen a clearly marked pit of the rete and the horny layer, which at this point has about ten to twelve times the normal thickness. Below this epidermal pit there is in the papillary body a dense lymphocytic infiltration, which is sharply demarcated from the lower part of the cutis. The infiltration decreases towards the margins of the slide. In sections stained with methyl-violet a substance is seen stained red metachromatically, in the papillary body, especially in the tips of the papillæ, in the form of smaller or larger clumps, and trabeculæ mostly grouped. The metachromasia, shape, grouping, and position in the skin is characteristic of amyloid. A second biopsy in the healthy skin in the vicinity of the diseased part shows no amyloid."

I diagnosed it as lichen obtusus, meaning by that not the type which is also called prurigo nodularis, but the other type, which is thought by many to be a form of verrucose lichen planus. At a meeting of the Section in December, 1921,[1] I showed an almost similar case, but there was certain evidence—apparently absent from this case—of lichen planus inside the mouth. The present patient had been treated in the out-patient department for some time with, apparently, no effect at all, though some X-ray treatment relieved the itching. Dr. Freudenthal saw the case and

[1] *Proceedings*, 1922, xv (Sect. Derm., 13).

suggested that it might be one of lichen amyloidosus. It is almost identical with a case which he pictured in his paper published two years ago.[1]

The question is whether this is a distinct condition, or a form of lichen planus verrucosus, with secondary amyloid change. Dr. Freudenthal does not find amyloid change in ordinary verrucose lichen planus, but he finds in these cases amyloid change in the skin of other parts, as well as in the lesions, though so far he has not done so in this case. Subsequent investigations have shown amyloid in the apparently normal skin of this patient.

Photograph taken before dietetic treatment.

Since the diagnosis of amyloidosis was made the patient has been put on a low protein diet (19½ grm. per diem) and the nodules and plaques have definitely flattened down somewhat.

Discussion.—Dr. MUENDE said he thought this case represented hypertrophic lichen planus. Dr. Freudenthal had said that amyloid change was not found normally in lichen planus, but he (the speaker) thought it was found in hypertrophic conditions of the epidermis, such as warts, and therefore it would not be exceptional to find it in the hypertrophic epidermal condition which Dr. Gray was showing.

1 *Archiv f. Derm. and Syph.*, 1930., clxii, 40.

Dr. A. M. H. GRAY (in reply) said he admitted that the question which had been raised was a difficult one. He thought this was a case of lichen planus hypertrophicus, but the two points he had mentioned had some bearing on the diagnosis. Dr. Freudenthal had pointed out that amyloid change did occur in many epithelial degenerating conditions, such as epithelioma and warts, and he appeared to hold the view that there were different types of amyloid disease of the skin, that in the case of epithelial tumours it was a pure degenerative change, and that the present type of case was a definite amyloid disturbance of the skin as a whole. And the fact that these changes did not occur in the ordinary characteristic warty lesions associated with lichen planus suggested that there might be a distinction between this type and ordinary warty lichen planus. But Dr. Freudenthal did not wish to lay down the law about it; he treated the cases as matter for investigation.

With regard to the white spots in the mouth, one morning there had been a crop of white spots on the inner side of the right cheek, but an hour later, when Dr. Freudenthal saw the case, practically all the spots had gone—that did not look as if they were lichen planus lesions in the mouth. One must await further observations on the subject.

Clinical Section

President—BERNARD MYERS, C.M.G., M.D.

[*March* 9, 1934, *continued*]

Two Cases of Vaquez's Disease.—GEOFFREY EVANS, M.D., F.R.C.P.

I. A man, aged 53, suffering from polycythæmia. First seen in January 1929, on account of chronic laryngitis. At that time he had a florid complexion suggesting Vaquez's disease, marked dental sepsis, enlargement of the liver down to the umbilicus and enlargement of the spleen to three fingerbreadths below the costal margin.

Blood-count.—R.B.C. 6,880,000 per c.mm.; Hb. 110%; W.B.C. 6,000 per c.mm. Differential count normal. Blood Wassermann reaction positive. Heart, blood-pressure, lungs, and urine normal.

The infected teeth were extracted, and the patient gave up alcohol; he had been in the habit of drinking both beer and whiskey. He was treated first with mercury and potassium iodide, and then with a course of intravenous arsenic. He has attended the out-patient department for the past three years, during which time he has continued at work, and has improved in health and strength.

Treatment has been with mercury and potassium iodide by mouth. In August 1932, he began taking phenyl-hydrazine 0·05 grm. twice a week, and continued to take this regularly until April 1933, when he developed jaundice and the drug was stopped.

In July 1933, he was put on a high-fat diet, namely, about 180 grm. daily. Since taking this high-fat diet he has felt better than he was when under any previous treatment.

The blood-count has never been very high :—

October 1932	Hb. 120%
February 1933	Hb. 130%
March 1933	Hb. 120%
July 1933	Hb. 108%

The red-cell count has generally been between 6,000,000 and 8,000,000.

October 1932	R.B.C. 3,960,000 per c.mm.
March 1933	R.B.C. 8,400,000 per c.mm.
July 1933	R.B.C. 9,960,000 per c.mm.

The case is shown from the point of view of the possible value of a high-fat diet in the treatment of polycythæmia.

It will be noted that there has been no change in the blood-count as a result of the diet. In Vaquez's disease there are, however, periods during which there is an exacerbation of symptoms and in which vascular complications may occur, and there are also periods of quiescence of symptoms with a feeling of better health. These variations in the course of the disease may occur without any recognizable change in the blood-count.

II. A woman, aged 50, was first seen in October 1931, complaining of recurrent epistaxis, shortness of breadth, and asthenia. Epistaxis was first complained of two and a half years previously. The striking features of this case were the pallor of the face, the blue-black colour of the lips, the plum-coloured tongue, and the spider telangiectases scattered over the face (especially the forehead), neck, arms, hands, fingers and upper part of the chest.

Blood-count.—R.B.C. 7,525,000 per c.mm.; Hb. 140%; W.B.C. 6,800 per c.mm. Differential count normal. Liver normal in size; spleen not palpable.

The case is shown as an example of "white polycythæmia" and also on account of the association of polycythæmia with telangiectasia.

This patient also has been on a high-fat diet for the past year, and this, combined with one venesection in June 1933, seems to have been of some therapeutic value.

So-called "white polycythæmia" is rare, and the association of telangiectasia with polycythæmia is particularly interesting. The association in this case is very close because the spider telangiectases completely, or almost completely, disappeared in a remission thought to be due to treatment by venesection and high-fat diet.

Dr. PARKES WEBER said he was especially interested in this case, since it showed a telangiectasia of the skin and mucous membranes, resembling that of Sir William Osler's familial telangiectasia, combined with erythræmia of the Vaquez type, though there was no obvious splenomegaly. Hepatic cirrhosis occurred in some cases of erythræmia (he had not long ago described such a case,[1] with the post-mortem examination after fatal hæmatemesis), and occasionally the telangiectases in cases of hepatic cirrhosis might simulate those characteristic of Osler's telangiectasia.

Advanced Pulmonary Tuberculosis with Tuberculous Laryngitis, successfully treated by Thoracoplasty and Cauterization of Larynx.—PHILIP ELLMAN, M.D.

B. K., female, aged 30, typist.

19.10.29.—Notified as a case of pulmonary tuberculosis, with extensive fibrocaseous disease and cavitation in left upper zone, scattered infiltration in right, and advanced tuberculous laryngitis. She then weighed 6 st. 9 lb. Afebrile. Mucopurulent sputum, a teaspoonful in twenty-four hours, with numerous tubercle bacilli.

January 1930 to September 1931. Sanatorium treatment. In 1931, artificial pneumothorax attempted, but unsuccessful.

2.9.31.—Left sanatorium at her own request. Prognosis regarded as bad. In view of this and of the fact that the disease was mainly unilateral and of the fibrocaseous type, the advisability of a thoracoplastic operation was considered. Examination of the larynx at that time showed thickening, irregularity, and infiltration of both vocal cords.

Skiagram, 2.10.31 (Dr. Stanley Melville).—Heart and mediastinum displaced to right. Right lung: emphysema, infiltration, and fibrosis of upper zone. Left

[1] *Proceedings*, 1934, xxvii, 1010 (Sect. Dis. in Child., 48).

lung: fibrosis and excavation of upper zone. Infiltration of lower zone with pleural involvement.

Skiagram, 12.7.32.—Left lung showed no change, but there was some further infiltration of right upper zone.

August 24, 1932.—Preliminary left phrenic evulsion (Mr. Tudor Edwards).

September 19.—Thoracoplasty completed in two stages (first stage September 5). Patient discharged on October 13, when sputum was negative. She was afebrile, and the chest wall had collapsed well. Mr. F. C. Ormerod completed the cauterization of the larynx in November 1932.

In 1933 she was again for six months in a sanatorium. She now weighs 7 st., and has no cough or sputum; her appetite is good and she feels well. Mr. Ormerod reports that the vocal cords are now normal in appearance; there is still a little infiltration in the inter-arytenoid space, but much less than there was. Argyrol is being injected fortnightly.

There is good collapse of the lung with some emphysema in the right. Voice not yet returned.

Despite the advanced lesion, complicated by tuberculous laryngitis, the patient has benefited considerably by the treatment. She is now being transferred to a tuberculosis colony, where she is going to continue her work as a typist.

Advanced Unilateral Pulmonary Tuberculosis successfully treated by Thoracoplasty. (Previously shown October 1931.[1])—PHILIP ELLMAN, M.D.

C. L., female, single, aged 32, clerk, notified as a case of pulmonary tuberculosis (sputum T.B. +) in 1919; had left-sided pneumothorax induced in 1928 for extensive infiltration of left upper and middle zones of left lung with cavitation of left upper zone. Right lung showed no evidence of tuberculous infiltration. After the pneumothorax, patient had neither cough nor sputum and general condition improved; weight then 8 st. Refills continued until February 1930, when, owing to pleural adhesions, they were discontinued.

Patient soon began to lose weight. Cough with blood-stained expectoration developed, tubercle bacilli being present in moderate numbers. The physical signs in the left lung were those of extensive cavitation of the left upper lobe with evidence of a thickened pleura at the base.

X-ray examination of chest (Dr. Stanley Melville): Heart, mediastinum and trachea displaced to left side; diaphragm on left side immobile. Left lung showed excavation of upper zone and considerable fibrosis and pleural involvement. Right lung showed compensatory emphysema but no infiltration.

Since the lesion was unilateral and showed marked evidence of fibrosis, good collapse of the left lung was essential for any progress. Mr. Tudor Edwards performed left thoracoplasty.

Operation.—2.7.30: Left phrenic evulsion. 23.7.30: Upper stage extrapleural paravertebral thoracoplasty. 6.8.31: Lower stage extrapleural paravertebral thoracoplasty.

September 1930 to March 1931: Sanatorium treatment. Uninterrupted progress.

Present condition.—No cough; trace of sputum, persistently negative.

Last examination: 13.2.34. No dyspnœa. Very good appetite. Some compensatory emphysema of right lung. She is now at full work, is completely afebrile; pulse normal; is in excellent health and weighs 9 st. 4 lb. (7 st. 8 lb. in May 1930).

1 *Proceedings*, 1931, xxv, 18 (Clin. Sect., 12).

Cardiac Aneurysm following Myocardial Infarction and Coronary Thrombosis (with Autopsy Findings).—PHILIP ELLMAN, M.D.

Case shown in November 1932.[1]

History.—A. J. C., male, aged 62, had had to give up work seven years previously because of attacks of constricting substernal pain and dyspnœa on exertion. No history of rheumatic or syphilitic infection.

On August 4, 1932, while comfortably seated in a train, he sustained a sudden and severe attack of constricting pain across the chest, radiating up into the neck and down the left arm. It remained acute for eight hours, and passed off very gradually after nearly two days' duration. During the acute attack of pain his temperature was 101°, and he suffered from severe shock.

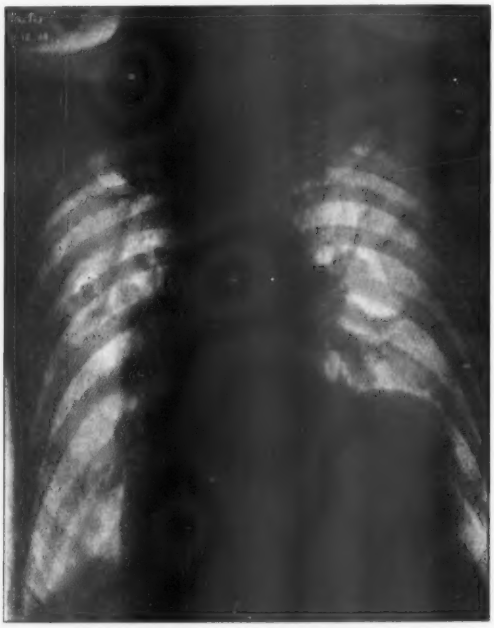

FIG. 1.—Aneurysmal dilatation of the left ventricle, 11.12.33, that is *one day before death.* Note the marked increase in size of the aneurysm since the skiagram taken on 20.10.32 (*Proceedings*, 1932, xxvi, 140 (Clin. Sect., 16)). Cf. X-ray and P.M. appearance of cardiac aneurysm in fig. 2.

On examination.—Heart: the apex beat was 4½ in. from the middle line; rhythm regular. Distant heart sounds. Aortic second relatively accentuated. No murmurs. Lungs: signs of fibrotic change at apices (old fibroid phthisis). Blood-pressure 105/70; subsequently it was still lower, and in November, just before death on December 12, 1933, it was 75/45.

Since the original attack of pain he had several recurrences of a similar constricting pain lasting several hours, with breathlessness.

An electrocardiogram showed low-voltage curves in all leads, left-sided axis deviation, split QRS-waves and flat T-waves in all leads. Serial electrocardiograms

[1] *Proceedings*, 1932, xxvi, 139 (Clin. Sect., 15).

taken subsequently at monthly intervals show no gross deviations from the original changes described, except that on one occasion there was definite evidence of a right bundle-branch block, but an electrocardiogram taken one month later assumed the features originally outlined.

X-ray examination showed limited diaphragmatic movements especially on the left side, heavy root shadows with old apical infiltration. Heart definitely increased in transverse diameter. Marked enlargement of left ventricle, lower border not defined. Obvious deformity of left ventricle above cardiac apex, compatible with aneurysm of left ventricle following coronary thrombosis with myocardial infarction (fig. 1). No visible pulsation seen, presumably owing to organized clot in aneurysmal

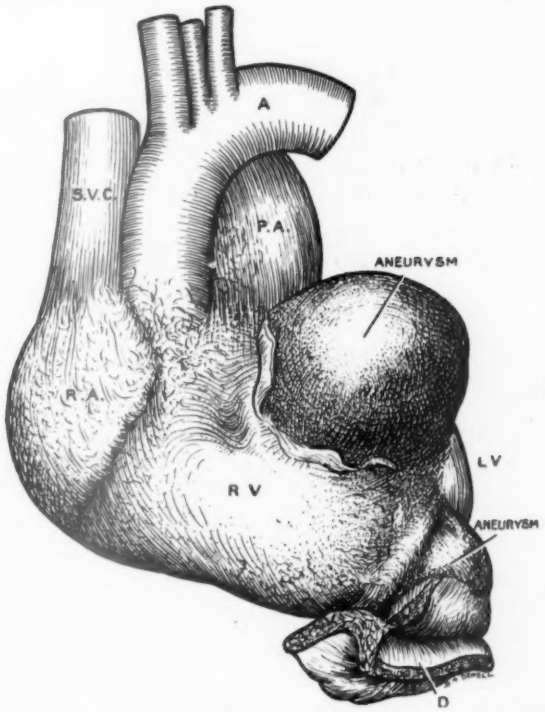

FIG. 2.—Photograph of post-mortem specimen of the heart. A, aorta; D, diaphragm. L.V., left ventricle; P.A., pulmonary artery; R.A., right auricle; R.V., right ventricle; S.V.C., superior vena cava.

sac. Serial skiagrams have shown a progressive increase in size of the aneurysmal dilatation. The patient died gradually of congestive heart failure.

It should be added that had the lower limits of the left ventricle been visualized on the screen by distending the stomach with gas another small apical aneurysm—seen in the autopsy specimen—would certainly have been seen.

Autopsy findings.—Lungs markedly congested; marked thickening of the pleura; apical tuberculous fibrosis confirmed.

The heart is greatly enlarged (fig. 2). In the upper part of the anterior wall of the left ventricle is a sac 7 by 8 cm. at its base, about 5 cm. in depth, growing upwards

forwards and to the left, but mainly forwards. It is adherent to the pleura and surrounding lung, and opens into the left ventricle through an aperture in its anterior wall near the septum. The cavity is filled with organized clot. The wall of the aneurysm is very thin, and appears to consist almost entirely of pericardium and adherent structures. The ventricular muscle surrounding the orifice of the aneurysm is composed of dense white fibrous tissue, i.e. old infarct. At the apex is a second smaller sac about 4 cm. in depth, and 5½ cm. in circumference. The thin wall of the sac appears to be composed mainly of fibrous pericardium, and the cavity is filled with organized clot. The aneurysm is pointing downwards and to the left, and is adherent to the diaphragm. This aneurysm was not visualized on the screen, since the lower limits of the left ventricle were not defined by distending the stomach with gas. The left coronary vessel was relatively small and was opened in the first few centimetres of its course. It was narrowed and atheromatous, and disappeared into the aneurysmal sac. No thrombosis was visible between the aorta and the origin of the sac. The right coronary vessel was patent, and larger than the left, its walls being smooth.

Whilst cardiac aneurysm is a fairly common lesion met with at autopsy, its recognition *in vivo* is not common.

I am indebted to Dr. Bedford and Dr. Keith for their help on the pathological side.

[In addition to the illustrations reproduced here, serial skiagrams, and electrocardiograms, and a drawing of the post-mortem specimen of the heart (posterior view), were shown at the meeting.]

Chronic Localized Bronchiectasis at Left Apex.—J. W. E. CORY, M.D.

H. M., aged 16, second son of a farmer, was seen at his home on June 26, 1933. He had had a cold a month previously, and was noticed to have been running an evening temperature ever since. He felt perfectly well and said he would carry on with his normal work if not prevented from doing so.

Condition on examination.—Pulse 96; temperature 100·2° F. Tongue slightly coated; teeth good; no obvious glandular enlargement in neck, axillæ, groins or abdomen. Area of cardiac dullness and sounds normal; no alteration of percussion note discovered in chest; auscultation also normal. Tonsils natural; maxillary antra and frontal sinuses equally translucent. Movements of limbs free and normal in all directions. Weight 7 st. 13 lb. Urine 1020, acid; no albumin; no sugar. Sputum: No tubercle bacilli discovered. Widal reaction negative to *B. typhosus*, *B. paratyphosus* A and B, and *B. abortus*. W.B.C. 21,200; polys. 86·5%; eosinos. 0·5%; lymphos. 13·0%.

12.7.33.—Admitted to West Suffolk General Hospital for further investigation as he continued to run temperature to 100° F. each day at 2 p.m.

W.B.C. 42,200; polys. 93%; eosinos. 1%; lymphos. 6%. Still no obvious physical signs, but breath sounds at left apex were diminished, and a short mitral systolic bruit was discovered.

X-ray examination of chest (Dr. Loughborough): "Left upper ribs retracted and an area of irregular density in left upper lobe. This part of the chest shows no expansion on respiration. Some dilated bronchioles are visible in the dull area." Conclusion: Fibrosis of upper lobe with possible bronchiectasis. Lipiodol might help.

During August a slight dorsal scoliosis to the left was noticed and as the boy continued to run a temperature, in spite of rest out of doors, creosote capsules were given by mouth and by inhalation. I saw him early in September with Dr. G. S. Haynes (Cambridge) who agreed as to the diagnosis and suggested a continuation of

the outdoor treatment and daily injections of transpulmin intramuscularly for a fortnight.

By mid-October the temperature had settled. X-rays still revealed the same area of irregular density at left apex. Breathing exercises were initiated.

29.12.33.—W.B.C. 24,000 : polys. 87·5% ; eosinos. 0·5% ; basos. 0·5% ; lymphos. 11% ; hyals. 0·5%.

The patient nevertheless feels perfectly well ; he has never had any symptoms or sputum.

22.2.34.—No rise of temperature since October. Weight 8 st. 10½ lb., i.e. a gain of 11½ lb. in eight months.

Dr. F. J. POYNTON said that it was an interesting coincidence that recently in his adult wards at University College Hospital, and also among his patients at the Hospital for Sick Children, Great Ormond Street, he had seen some most puzzling cases of massive dullness over the lower lobes of the lungs. In three cases this was accompanied by considerable fever. In one which was explored the needle penetrated a resistance of mossy consistence, but there was no pus. The course had been long and the massive dullness remained after the fever ceased. The radiographic appearances were striking. He had not yet convinced himself of the correct diagnosis in these cases but in one fatal case in a child, the upper lobe was solid, with a subacute pneumonic change, and subpleural small abscesses had finally infected the entire pleural cavity.

[*May* 11, 1934]

Physiological, Pathological, and Clinical Aspects of Diseases of the Gall-bladder

By CHARLES NEWMAN, M.D.

[ABSTRACT]

THE function of the gall-bladder—to store up and deliver bile in the manner most advantageous to the process of digestion—is conventionally divided into the motor and concentrating functions. Between meals the little terminal sphincter of Oddi in the tip of the papilla of Vater is closed by sympathetic innervation and bile flows into the relaxed gall-bladder where it is concentrated and stored. After a meal, vagal activity contracts the gall-bladder, which fills the fibro-elastic common duct, relaxes the sphincter, and sets the detrusor muscle of the ampulla into peristaltic activity, emptying the bile into the duodenum. Excessive vagal activity, which may be due to many causes, produces spasm of both gall-bladder and ampulla, and, as the ampulla is by far the strongest muscle in the system, the result is complete stoppage of the bile flow.

The abnormal physiological processes which lead to symptoms are very similar, whatever the disease producing them, and therefore the clinical pictures of diseases of the duodeno-biliary region tend to resemble one another.

Cholecystitis may be conveniently considered under three headings : (1) Acute cholecystitis, which is an acute abdominal emergency best treated by operation, although, when necessary, it can sometimes be successfully treated medicinally. (2) Chronic cholecystitis with gall-stones, which is a common cause of dyspepsia and which may be alleviated, but not cured, by diet and the administration of belladonna and bile-salt. (3) Chronic cholecystitis without stones, an uncommon

disease not to be diagnosed without thorough investigation, and never to be treated surgically without proof. A similar clinical picture can be produced by simple vago-spastic distension, by chronic appendicitis, and by duodenal ulcer—amongst a multiplicity of causes. True chronic cholecystitis is seldom, if ever, completely apyrexial and it leads to a flatulent type of dyspepsia. The duodenal tube is an invaluable aid to diagnosis ; it gives diagnostic results in cases of spastic distension, and examination of the bile is of great help in the diagnosis of cholecystitis. The common finding is multiple large cholesterol crystals ; the absence of pus and organisms is no evidence that the gall-bladder is not inflamed, as cholecystitis is essentially a subserous, not a submucous, inflammation.

The bacteriology of cholecystitis and the path of infection are unprofitable subjects of discussion. Originally the *Bacillus coli* was held to be the important organism, and after a temporary movement in favour of staphylococci and streptococci, there is now a return to the *B. coli* school. Opinion is likewise veering away from an intramural blood-stream infection, in favour of infection via the bile, with organisms excreted by the liver.

With regard to the pathology of gall-stones, the important point to remember is that all gall-stones are not of similar nature or similar causation, and that combination stones are common and represent a succession of ætiological factors in time. The important varieties of stone are : (1) The infective type, represented by the common faceted, the barrel, and the raspberry stones. They are all built on an organized nucleus and the last two types are simply overgrown nuclei of the faceted form. The cholesterol for their formation is provided by a disorder of concentrating function. The normal gall-bladder absorbs small quantities of the addition-compound with bile-salts, in which form cholesterol is held in solution in the bile. In a dializing sac or an inflamed gall-bladder the bile-salts are split off and pass out, leaving the cholesterol in insoluble form behind. Why the crystals form stones is another matter, probably connected with the protein-containing inflammatory exudate. The raspberry stone is the characteristic accompaniment of the strawberry gall-bladder, which is also usually due to inflammation. (2) The cholesterol solitaire is a radial stone without a nucleus, which is possibly due to metabolic disorder, possibly to an unidentified form of infection. It is rare and seldom causes symptoms. (3) The pigment calcium stone is associated with disorders of pigment metabolism and is remarkable for containing large quantities of iron and copper. (4) Some calcium carbonate stones contain copper and are probably related to (3), others are the result of cystic-duct obstruction which is the principal cause of the deposition of calcium carbonate in the gall-bladder. (5) The earthy stone is a loosely-formed deposit which can arise in the bile-ducts as well as in the gall-bladder, and is the characteristic formation round foreign-body nuclei.

The dyspeptic prodromal symptoms of gall-stones are caused by cholecystitis and not by cholelithiasis, and are therefore to be expected only in the case of infective gall-stones.

Carcinoma of the gall-bladder is not caused by gall-stones, but by chronic infection ; a third of the cases are found in stone-free gall-bladders. Where carcinoma and stones co-exist they are both due to the same cause—cholecystitis.

With regard to the clinical examination of the gall-bladder, Vincent Lyon's percussion sign is a valuable adjunct to Murphy's sign. The former means that there is something wrong with the gall-bladder, the latter that it is inflamed. Though it is a mistake to say that the gall-bladder can never be felt, what is taken for a palpable gall-bladder is often a rigid segment of the right upper rectus.

The Radiology of the Gall-Bladder.

By H. GRAHAM HODGSON, C.V.O., M.R.C.P.

I SHOULD like to preface my remarks on the radiology of the gall-bladder with a word of warning. I should like to remind you that radiology never was, and never will be, intended to take the place of routine clinical methods. It is of use, and often of the greatest use, in confirming and amplifying the deductions of the clinician, and occasionally the diagnosis may be based to a large extent on the X-ray appearances, but radiology only serves its real purpose when it is recognized as a useful ancillary branch of the medical forces and not as the spearhead of diagnostic attack. I say this advisedly, because I see an increasing and dangerous inclination among the coming generation training at our hospitals to take short cuts to a diagnosis by referring cases to the X-ray department without first making a thorough attempt to progress along the difficult but definite path of clinical examination. I am in no way depreciating my own subject by saying that where this path fades out is the point at which to bring in radiology, but not before.

After these preliminary remarks, I will try to demonstrate how far it is possible for the radiologist to assist in the diagnosis of gall-bladder pathology.

The radiology of the gall-bladder is divided into two distinct phases, the first extending from 1899 to 1924. In the former year the first X-ray plates demonstrating gall-stones were taken by Carl Beck in New York, Thurstan Holland being the first radiologist to take them in this country. During this period powerful X-ray transformers superseded the old-fashioned coil, and intensifying screens, double-coated films, and the Potter-Bucky diaphragm were introduced, all helping greatly towards a vast improvement in radiographic technique. But, in spite of more rapid exposures and more detail and contrast in the radiograms, the limiting factor in the utility of X-ray examinations for gall-stones was the calcium content of the stones, for unless this calcium content is sufficiently large gall-stones are not opaque to X-rays. Since only from 10 to 15% of gall-stones contain this sufficiency of calcium, the direct method, as it is called, is only of use as positive evidence, and of little value when no gall-stones are seen. Also, of course, it gives no guidance as to biliary function.

The most common type of stone visible by direct radiography has an opaque periphery and a trans-radiant centre, due to deposition of calcium on a non-opaque cholesterol calculus. They are much more commonly multiple than single and are frequently faceted. When they are incompletely covered by calcium, the calcified portion appears as a sickle-shaped shadow or segment of a circle. These shadows are sometimes confused with calcification in the lower rib cartilages. Radiograms taken at full expiration will usually clear up any doubt on this point, as the calcified portion of the rib cartilage will, of course, maintain its position relative to the rib, whereas the partially calcified gall-stone will not. A further distinguishing point is that calcification in a rib cartilage is never concave downward, whereas partial calcification in a gall-stone may be.

A less common type of stone is that with a bilirubin calcium nucleus and a cholesterol periphery.

A point that should be borne in mind is that radiography is based on relative, and not on absolute, opacity and that, therefore, a gall-stone examined by X-rays after removal may cast a shadow, but in the body its lime content may be too small to make it more dense than the surrounding tissues.

Apart from visualization of opaque gall-stones, the direct method only in rare instances gives any information as to the condition of the gall-bladder.

The gall-bladder is, of course, normally invisible by X-rays, but occasionally, when the walls are very greatly thickened by disease, its shadow can be seen.

Again, a barium meal examination sometimes shows sharp angulation of the second part of the duodenum toward the right, suggesting a pericholecystitis and adhesions.

Flattening of the duodenal cap may suggest adhesions to, or pressure by, a distended gall-bladder (fig. 1, Plate I). I have also seen two cases in which a barium meal examination revealed a fistula between the duodenum and the biliary tract (fig. 2).

Apart from such rare instances, however, and the visualization of a small percentage of gall-stones, the radiology of the gall-bladder up to 1924 was only of value in a small number of cases.

In 1923 Graham and Cole conceived the idea that, if the gall-bladder could be rendered visible by the roentgen ray, after being filled with a contrasting substance, not only might earlier and more certain signs of a pathological condition in that organ be made recognizable, but such a procedure might also be an accurate index of function. In 1909 Abel and Rowntree showed that the halogen derivatives of phenolphthalein compounds were largely excreted in the bile, and thirteen of these compounds have been shown to be of sufficient atomic weight to render the bile opaque to X-rays. Most of them had disadvantages, consisting of either too great toxicity or of general staining of the body tissues. Two of them, phenoltetraiodophenolphthalein and tetraiodophenolphthalein, were found to be much superior to the others as contrast media in cholecystography. Sodium tetraiodophenolphthalein is the one most commonly used to-day.

With regard to toxicity, the lethal dose given intravenously is 2·7 grm. per kilogram of body weight, which is from five to seven times the dose used in cholecystography.

In the early days of cholecystography the majority of the examinations were carried out by the intravenous method, but this had the disadvantage of involving a minor operation, and if, owing to poor veins or unskilful injection, any of the solution got into the surrounding tissues, a possibly severe reaction might occur.

Owing to improvements in the preparation of the dye, the intravenous method has given place almost universally to the oral method, the former being reserved for those cases in which the stomach is peculiarly irritable and the patient cannot retain the dye. Kirklin tested the reliability of the oral method in 250 patients in whom it had failed to give a shadow. When the dye was later given intravenously to these patients, only 1% showed normal gall-bladder filling.

The essential point of oral cholecystography is that a sufficient quantity of the salt should be absorbed from the intestine. This may be prevented by vomiting or diarrhœa, and the dye should therefore be administered in such a manner as to avoid these difficulties.

Although in cholecystography substances opaque to X-rays are used, the underlying principle is entirely different from that of outlining a hollow viscus, such as the stomach or a bronchus. In cholecystography, and also in intravenous pyelography, one makes use of the specific function of a system and its selective action on a particular substance to engender a difference in density. Cholecystography, therefore, as Graham points out, is largely a test of physiological capacity, in contradistinction to the almost wholly mechanistic nature of the opaque meal examination.

For this reason it is essential for the radiologist to have a thorough understanding of the physiology as well as the anatomy and pathology of the biliary system.

Technique.—In cholecystography a careful and thorough technique is essential, not only in the radiography but also in the preparation of the patient and general conduct of the examination. Slight variations in technique to suit individual preferences may be made, but the main physiological factors governing the examination must be strictly adhered to. For instance, should the patient, unknown to the

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PLATE I.

FIG. 1.

FIG. 2.

FIG. 3.

FIG. 4.

FIG. 5.

FIG. 1.—Flattening of duodenal cap by a distended gall-bladder containing stones. Note two stones in common duct.

FIG. 2.—Fistula between first part of duodenum and common bile-duct after a barium meal. Note barium in all ducts and the biliary capillaries.

FIG. 3.—Gall-stone in apparently normally functioning gall-bladder.

FIG. 4.—Shadow of pancreatic calculus over upper right angle of second lumbar vertebra Note normal gall-bladder shadow.

FIG. 5.—Same case as fig. 4. Oblique view of stomach, showing calculus shadow between first and second parts of duodenum.

PLATE II.

FIG 6.

FIG. 7.

FIG. 8.

FIG. 9.

FIG. 6.—Dye-filled gall-bladder shadow superimposed on, and concealed by, shadow of spine.
FIG. 7.—Oblique view of same case as fig. 6.
FIG. 8.—Redundant neck of dye-filled gall-bladder simulating a gall-stone. Taken at full expiration.
FIG. 9.—Same as fig. 8, but at full inspiration, showing disappearance of rounded shadow.

radiologist, have a meal between taking the dye and the taking of the first radiogram on the following day, probably no dye would be found in the gall-bladder, and an erroneous conclusion of the pathology would be arrived at.

Forty-eight hours previous to the examination, the patient should take a purgative. On the following day a control radiogram should be taken. A large film, 15 in. by 12 in., should be used, so as to include the whole area from the right diaphragm to the right iliac fossa. It should also include the lumbar and lower dorsal spine. At least two radiograms should be taken, with varying penetration, so as to obtain the optimum result according to the radiographic density of the patient. The patient should be carefully instructed to hold his breath at full expiration, and the exposure should be as short as possible—not more than a second. The slightest movement due to respiration or to peristaltic action in the adjacent bowel will blur the faint outline of a calculus so as to render it invisible. In order to avoid distortion of the gall-bladder outline, the tube should be as far away from the film as is consistent with a rapid exposure. I usually employ a four-foot distance. This radiogram should be carefully examined, not only for shadows of opaque biliary calculi, but also for renal calculi or any pathological condition of the spine which might give rise to symptoms in the right upper quadrant. In the evening the patient takes a meal, and it is important that this meal should be prescribed, as it *must* be free from fat. If fatty foods are allowed at this meal, a large number of normal gall-bladders will fail to fill, or show poor filling. Half an hour after the meal the dye is taken. From 3·5 grm. to 6 grm. of sodium tetraiodophenolphthalein may be given, according to the patient's weight. The dye used to be taken in gelatin or keratin capsules, but these frequently failed to dissolve, and most workers now give it mixed with grape or orange juice. Rowden gives one-sixth of a grain of morphia in a tablet placed under the tongue. This very largely prevents vomiting and retards the action of the bowels, thereby helping absorption. After taking the dye the patient must take nothing to eat or drink until told to do so. The first radiogram should be taken on the following day on a fasting stomach, fourteen hours after the ingestion of the dye. The radiographic technique required is of the highest order. A Potter-Bucky diaphragm is generally used in order to give greater contrast, and, since the gall-bladder may be situated anywhere between the tenth rib and the iliac crest, the first film should be a large one. The patient should wait until the film is developed and, unless it shows the utmost possible detail, it should be repeated until the best radiographic factors of current and penetration, according to the thickness of the body, are arrived at. At fourteen hours, in a normal case, a distinct and definite gall-bladder shadow should be visible, owing to commencing secretion and concentration of the dye. I take further radiograms at sixteen, eighteen, and sometimes twenty, hours. During this time the patient is fasting, the sphincter of Oddi is closed, and the dye consequently passes into the gall-bladder and is there concentrated and becomes opaque to X-rays. The maximum degree of concentration is usually reached at from eighteen to twenty hours. At one of these times radiograms are taken at full inspiration and full expiration, to see whether the gall-bladder moves normally with respiration. A meal containing a high proportion of fat is then given, the best medium for this purpose being an egg beaten up in milk. Up to this time the stomach has been empty, no contractions have been taking place in the duodenum to aid the flow of bile, the sphincter of Oddi has been closed, and the gall-bladder has consequently become distended with concentrated dye and bile. Very shortly after the ingestion of this meal, the biliary neuro-muscular mechanism responds to the stimulus. A radiogram taken an hour subsequent to this meal shows a dense, but smaller shadow. Whether this diminution in size is the result of decreased common-duct pressure and relaxation of the cystic elastic fibres, or whether it is due to contraction of the muscularis of the gall-bladder, is still probably debatable, from the purely physiological point of

view, but all that matters from the radiological standpoint is that the gall-bladder shadow contracts and becomes denser. Contractions are now taking place in the duodenum, the valve of Oddi is patent, and a free flow of bile is taking place into the duodenum; first of all the non-opaque common duct bile, then the dye-stained concentrated cystic bile, and finally the thin hepatic bile. At this stage, between two and four hours after the ingestion of the fatty meal, the gall-bladder shadow is again invisible.

From this short description it will be seen that cholecystography is essentially a test of function.

The gall-bladder may have been diseased in the past and have recovered. It may even contain gall-stones (fig. 3), which may result either from past infection or stasis, and still function in the normal manner which I have described. On many occasions, when examining a patient for a lesion of the gastro-intestinal tract, urinary tract or spine, I have discovered gall-stones by accident where there were no symptoms suggestive of gall-bladder pathology. On several of these occasions I have carried out a cholecystographic examination and found the gall-bladder secretion concentrate the dye normally. Normal functioning, as shown by cholecystography, indicates that a sufficient quantity of the dye has been absorbed into the blood-stream from the intestine, that the liver is excreting the dye normally, and that the mucous membrane of the gall-bladder and its vascular and lymphatic systems are functioning normally. It also indicates that the walls of the gall-bladder are capable of contracting normally on the stimulus of a fat-containing meal and expelling its contents.

Such is the behaviour of a normal cholecystogram. But, apart from the demonstration of a normally functioning gall-bladder, the outlining of the gall-bladder by the dye is not infrequently of use in identifying shadows which might otherwise be thought to be in the gall-bladder. Here are two calculi which, from their laminated and faceted appearance were at first thought to be in the gall-bladder, but a cholecystogram showed them to be renal calculi. Again in this case, the visualization of a normally filling bladder was of great use (fig. 4). The patient had had a sudden and severe attack of what was thought to be biliary colic. He lived in a somewhat inaccessible part of the Midlands, and his doctor asked me to send down the dye and come and make a cholecystographic examination on the following day with a portable X-ray apparatus. The fourteen-hour radiogram shows a normally filling gall-bladder, but there is this shadow here. It is solitary and its transverse position suggested to me the possibility of its being in the duct of Wirsung. On examination the urine showed some sugar, and there was some undigested fat in the stools, so I then gave the patient a barium meal and took a radiogram in the lateral position, thus outlining the duodenum, and showing this pancreatic calculus to be apparently about half an inch from the ampulla of Vater (fig. 5).

Turning next to the pathological gall-bladder and its cholecystographic appearances: In the first place, if there is no visible gall-bladder shadow throughout the examination, if the films are technically perfect, if the patient has not vomited the dye or had severe diarrhœa, then this non-visibility is indicative of a pathological condition. I am taking it for granted that the whole area from the tenth rib to the ilium has been covered, as the gall-bladder shows as wide a latitude in position as the stomach, and may even be situated to the left of the lumbar spine as in cases of situs transversus. Its shadow may also be superimposed on that of the lumbar spine and thereby hidden, unless lateral views are taken (fig. 6, Plate II). Unless one examines this film very closely, one might be inclined to say that no shadow was present, but the border of an abnormal shadow is seen here and, on turning the patient into the lateral plane (fig. 7), a normally filled gall-bladder is seen, situated abnormally in front of the lumbar spine.

When, however, after these precautions no gall-bladder shadow is found, its absence indicates either: (1) that the liver is not excreting the dye, (2) that the

cystic duct is blocked, or (3) that there is a breakdown through disease of one or more of the links in the concentrating chain of the gall-bladder, that is, either of the mucous membrane or of the vascular or lymphatic drainage.

Chronic cholecystitis is by far the most common cause of non-visualization of the gall-bladder. This non-visualization is due not to failure of the dye to enter the gall-bladder, but to failure of the gall-bladder to concentrate the dye and so make it opaque to X-rays.

There are also causes extrinsic to the gall-bladder. As Brailsford has pointed out, in cases of active gastric or duodenal ulceration there may be non-visualization of the gall-bladder. Whether this is due to reflex spasm of the gall-bladder, to dysfunction of the sphincter of Oddi, preventing filling, or due to interference with concentration as a result of hyperæmia, I do not know, but it is a factor to be taken into account, and suggests the advisability of a barium meal examination of the stomach and duodenum, before concluding that non-visualization of the gall-bladder is due to intrinsic disease of the biliary tract.

Faint visualization.—In this connection, great caution has to be exercised and many factors taken into account.

In the first place, "radiology is a study of contrasts in density," and density of the gall-bladder shadow is relative to the density of the surrounding parts. In a thin hyposthenic patient, a normally concentrating gall-bladder casts a fairly dense shadow, whereas, in a heavily built, hypersthenic patient, an equally normal gall-bladder, situated high in the right upper quadrant and partly covered by the lower margin of the liver, casts a shadow much less dense. Again, there is a fairly wide variation within normal limits of the concentration and consequent density of the dye in patients of similar body weight and habitus. Therefore all these factors have to be very carefully weighed before we come to the conclusion that there is inadequate concentration of the dye and consequent disease of the gall-bladder, resulting in faint visualization.

Gall-stones.—Radio-opaque gall-stones, as I have mentioned before, should be visualized in the control radiograms, taken before the administration of the dye on the day previous to the examination. When, however, the patient has been unable to attend on the previous day, one should beware of confusing shadows such as this (fig. 8). This is not an opaque gall-stone, but the shadow of the dye in a redundant neck, superimposed on the shadow of the upper part of the gall-bladder, as is shown by this radiogram (fig. 9) taken a few minutes later, at full inspiration instead of full expiration.

Non-opaque, pure cholesterol stones are shown as negative shadows due to the filling defect they cause in the dye shadow.

Gas-shadows in the small gut, when superimposed on the gall-bladder shadow, sometimes cause difficulty by simulating gall-stones, but radiograms taken at full inspiration and full expiration show these gas-shadows and the gall-bladder shadow to move unequally.

With a faint gall-bladder shadow, gall-stones may show only very faintly, or not at all, in the prone position. Some slides which Dr. Bush has kindly lent me show beautifully how this difficulty can be overcome by examining such cases in the erect posture. By placing the patient in the erect position a much denser shadow is obtained, the gall-stones having gravitated to the lower pole.

The same method can be applied to cholecystography. A poorly filling gall-bladder may show no calculi in the prone position, not because they are not present, but because the inadequately concentrated dye and the gall-stones are spread throughout the viscus, while the patient is lying down, and they do not present a sufficient contrast in density to throw the gall-stones into relief. But when the same patient is placed in the erect posture, both the dye and the gall-stones are collected in the lower pole, and the stones become immediately visible.

Occasionally one sees cases in which the gall-bladder secretes and concentrates the dye but is abnormally slow in evacuating its contents. If this delayed evacuation has been preceded by a normally concentrated shadow at fourteen hours, one has to consider the implications very carefully before coming to the conclusion that the delayed emptying is caused by disease and thickening of the gall-bladder walls, interfering with normal contraction. For it must be remembered that, in order to visualize the gall-bladder at all, the dye has to be not only secreted, but concentrated, and it would be the least probable solution that a gall-bladder should have walls capable of concentrating the secreted dye in a normal manner, but incapable, through disease, of contracting and expelling its contents. Therefore in such cases one should first search for a more simple solution. If control radiograms have not been taken previous to the administration of the dye, such a shadow may be due to a gall-bladder with walls so thickened as to themselves cast a shadow, or bile sand may cast a shadow indistinguishable from a dye shadow. If, however, such possibilities have been excluded by control radiograms in such cases, the probable explanation of the persistent shadow is re-absorption of the dye from the colon. After these primary considerations of the ætiology of slow emptying, one takes into account the possibility of a gall-bladder which has been over-distended by stasis, as in multipara, and is consequently sluggish in contraction, but not necessarily infected. Also, in some instances, adhesions about the neck of the gall-bladder may admit the thin hepatic bile and unconcentrated dye, but retard the evacuation of the viscid and concentrated product.

Finally, cholecystography is a comparatively young subject, and there is still a great deal to be learnt, but as Graham points out in assessing its value, symptoms from an organ are always due to functional disturbances, although usually traceable to a pathological lesion.

Therefore, the method should not be condemned if, on the one hand, a gall-bladder shown by it to have normal functions, exhibits, at operation, a few old adhesions, representing some healed process, or on the other hand, a gall-bladder may appear macroscopically normal at operation in spite of a complete failure of concentrating function.

Affections of the Gall-bladder.

By A. James Walton, M.S., F.R.C.S.

Affections of the gall-bladder necessitating surgical treatment may arise from changes in the gall-bladder itself or be due to a primary lesion in the ducts below. They may be considered under the following headings.

Cholecystitis without stones.—Although inflammation of the gall-bladder is the common precursor of gall-stones, patients do not, as a rule, seek surgical advice until the stones are formed. In the past, when typhoid fever was a relatively common disease, an acute suppurative cholecystitis was not infrequently found during the course of the fever. It had existed for so short a time that stones had not yet had time to form and yet the acute inflammation demanded surgical interference. To-day such cases are rare and should only be diagnosed with a short history of acute inflammation in the gall-bladder area which has occurred in the course of an acute infection elsewhere.

The necessity for operation in such cases will depend upon the urgency of the symptoms. If the condition is mild and can be regarded only as an acute catarrhal inflammation, the patient may be watched, in the hope that the acute symptoms

will subside, although it is true that even with such a lesion gall-stones will probably form later. If the condition is progressive, operation must be undertaken. The ideal treatment is, without question, cholecystectomy, for acute suppurative cholecystitis is never completely cured by medical means, and even if the gall-bladder be drained stones will probably form at a later date. If, however, the patient is profoundly ill, it may be necessary to be content with cholecystostomy as a life-saving-measure.

Chronic cholecystitis is much too often diagnosed to-day in the absence of gall-stones, and what is of greater importance, the patient is much too often subjected to operation. Not only is chronic cholecystitis the common precursor of calculi, but probably no case of chronic cholecystitis could exist for long without such formation of stones. If no stones are present there is, however, a small possibility that the infection may be cured by medical treatment, and the risks arising from such a lesion are so relatively slight that there is hardly ever any indication for urgent operation. If medical treatment has been given a fair trial and has failed, stones will almost certainly form, and there can be little excuse for the growing practice of performing cholecystectomy upon incomplete evidence of cholecystitis, as a method of cure for remote conditions due to an imaginary infection of this viscus.

A possible exception is the interesting condition known as a strawberry gall-bladder, a change in which there is a collection of lipoid substance in the apices of the mucosal folds. It only occurs in chronic cholecystitis and is often associated with calculi, although occasionally it may be seen with such conditions as eclampsia. The symptoms are those of a long-continued chronic cholecystitis which has failed to react to medical treatment. At operation there are generally evidences of chronic change, such as opacity and thickening of the gall-bladder wall, but occasionally it can only be recognized after the gall-bladder has been opened. Cholecystectomy is probably always required to produce a cure.

Gall-stones.—It may now be accepted that the common variety of stones is always due to chronic inflammation, and that the stones are formed by a process of adsorption around an irreversible colloid, and thus are insoluble. Rare exceptions to this method may be the pure pigment stones which are found with blood-destroying diseases such as acholuric jaundice and malaria. There is a certain amount of evidence that such stones are simply deposited as masses of pigment from a super-saturated solution and thus may be again absorbed, but even in such cases the presence of the stones soon leads to a secondary cholecystitis with deposits of cholesterin and calcium salts, which are unabsorbable. I am showing a specimen which, so far as I know, is a unique example of stones, the greater part of which were formed of pigment and had been absorbed, but portions of the unabsorbable cholesterin coats are left, forming structures which resemble extremely hollow and decayed teeth. Pure cholesterin stones are often regarded as metabolic in origin, but, to my mind, the evidence that they are secondary to an inflammatory change is overwhelming, for they are always amorphous, they are never formed in the ducts, and in my own experience they have always been associated with inflammatory changes in the bladder-wall.

It is, therefore, manifest that, with the possible exception of the pure pigment varieties, medical treatment can never be expected to dissolve the calculi. Methods of accomplishing this are often advocated, and I have one case in which a patient had symptoms of gall-stones and X-rays showed 15 stones in the gall-bladder and one in the cystic duct. The patient had a course of medical treatment with various oils in a nursing home, and was shown 18 masses, said to be the gall-stones which he had passed per rectum during the treatment. He went home greatly relieved, but next day had an attack of biliary colic; a further X-ray examination revealed 15 stones still in the gall-bladder and one in the cystic duct. I operated on this

patient, and he now has 16 stones in a box and no biliary colic. I may say that in a series of over 800 cases of operation on the gall-bladder I have not, as yet, seen a single example of chronic cholecystitis in which it could be fairly assumed that all the stones had been passed down the ducts.

Therefore, the most that can be expected of medical treatment is that it may keep the symptoms of stones in abeyance so that the patient may be given a comfortable, if restricted, life. The chances of this are small, but it may be tried in patients who are able and willing to live a sheltered life, and more especially in those who are bad operative risks. On mechanical grounds, the only curative treatment for gall-stones is operation, but often these patients are very stout and in other ways are bad subjects, so that the dangers of operation must be carefully balanced against the discomfort and the possible complications which may arise from the presence of the calculi. Manifestly, a live patient with gall-stones is better than a dead patient with no stones. In bad subjects medical treatment, even if unable to make the patient's life safe and comfortable, may do much to diminish the risk of subsequent operation, but it must not be continued too long, for the onset of complications—such as long-continued colic, the presence of acute cholecystitis, impaction of the stone in the common bile duct, or of carcinoma of the gall-bladder—endanger the patient's life and add greatly to the risk of operation.

Therefore the chief indications for operative interference are the presence of repeated attacks of colic, the presence of attacks of subacute cholecystitis, and, more especially, the onset of transient or progressive jaundice. My own experience has led me unhesitatingly to take the view, and this in opposition to many, that the presence of acute cholecystitis is an urgent condition demanding immediate operation, that although some cases may quieten down if left, the dangers of expectant treatment are great and the risk of immediate operation is not greater than—if as great as—that of operation performed after several days. The same is true of jaundice. It is impossible to tell in which cases the jaundice will abate and the longer it is left, the greater is the danger, owing to the effects of back-pressure upon the liver.

Operative technique.—Perfect access can always be obtained to the gall-bladder by means of an upper right paramedian incision which gives the minimum of injury to the abdominal wall. Except in very ill patients the operation of choice should always be cholecystectomy, for with cholecystostomy it is almost impossible to be certain that stones are not overlooked and even if all are removed they may later be re-formed in the diseased gall-bladder. The removal of the gall-bladder should, whenever possible, be commenced by division of the cystic duct, for the dangerous area is then treated at the beginning of the operation when the field is unobscured by blood. If the gall-bladder is very tense, owing to an acute inflammation, access to the cystic duct may be facilitated by first aspirating the gall-bladder. In any case, however, the surgeon must make an absolute rule never to divide any structure until the three ducts—cystic, hepatic, and common bile-duct—are all exposed; otherwise the risk of injury to the common duct is very great. When this has been accomplished the cystic duct may be clamped and divided and the cystic artery then divided between ligatures. No operation for gall-stones can be regarded as complete unless the common duct is explored from within, but in very stout or ill patients who have never had jaundice the risk of overlooking stones in the common duct may be less than that of exploring this structure. In all others a pair of curved forceps should be passed down the duct and, if no stones are found, should be made to enter the duodenum through the papilla of Vater. The forceps are then opened to dilate the ampulla so that there is free drainage of the duct into the duodenum. The opening of the cystic duct is then closed and embedded, the gall-bladder removed, the raw area of the liver sutured, a drainage tube inserted down to the closed end of the duct, and the abdominal wall closed.

Carcinoma of the gall-bladder and ducts.—These conditions are not so rare as is frequently supposed (Table I). When arising in the gall-bladder, carcinoma is frequently associated with stones (Table II), and the condition is nearly always diagnosed as cholecystitis or as calculi. In some cases, indeed, the true condition is only suspected after the gall-bladder has been removed for stones and microscopic sections have been cut. These cases, naturally, are those with the best prognosis. In others the condition is manifest at operation, for unfortunately the disease rapidly spreads to the liver, where secondary deposits may be evident. The involvement may be by direct spread, or scattered secondaries may be seen which are probably conveyed by the lymphatics. In the former case an attempt may be made to bring about a cure by the performance of a cholecystectomy, together with a widespread resection of the adjacent portions of the liver, but in such cases the outlook is poor.

TABLE I.

	Total	Obstruction	Males	Females
Gall-stones	662	—	149	513
Stones in C.D.	133	—	27	106
Obstruction of C.D.	—	103	23	80
Recurrent stones :				
Gall-bladder	17	—	6	11
Common duct	11	11	4	7
Both	16	16	2	14
Reconstruction of C.D. ...	34	—	8	26
Injuries of C.D.	—	17	2	15
Carcinoma of G.B.	18	—	4	14
Carcinoma of C.D.	28	28	8	20
Carcinoma of pancreas ...	47	33	28	19
Chronic pancreatitis	61	35	30	31
	1,027	243		

TABLE II.

Carcinoma of G.B.	18
Without stones	12
Carcinoma of ducts	28
Without stones	21
Ampulla	9
Common duct	6
Junction	10
Hepatic duct	3

When the carcinoma arises in some portion of the duct, the diagnosis from stones is not always easy, although the clinical differences are supposed to be definite. A short history of constant and progressive jaundice leading to a green-grey colour, rather than the orange-red jaundice seen with obstruction by a stone, the absence of colic—or, if it is present, its lack of association in time with the onset of the jaundice—and the presence of a dilated gall-bladder are indications of an obstruction from carcinoma but in many cases the diagnosis is only settled at operation. In every case, however, an operation should be advocated as soon as the diagnosis of obstructive jaundice is made, for such a condition is certain to proceed to a fatal termination, and even if the obstruction be not removed, much relief may be obtained by palliative measures. An operation should be performed at the earliest date because, with the complete obstruction of carcinoma, the liver suffers severely and the secretion of bile soon ceases, so that the ducts become filled with a watery fluid, the so-called white bile. The presence of this in the ducts warrants a grave prognosis even if the patient appears to be in a very good condition at the end of the operation.

The pathological changes produced by carcinoma will vary according to the position of the growth. If at the papilla, the pancreatic and biliary ducts will be dilated and the gall-bladder will be distended with bile; if higher up in the ampulla, the pancreatic duct may escape; if situated at the junction of all three ducts, the

common duct will be collapsed and the gall-bladder will be dilated, but it will contain no bile; if situated above the insertion of the cystic duct, the gall-bladder also will be collapsed and empty. When the growth is situated in the first two positions it is usually only possible to perform a cholecystoduodenostomy, although it is occasionally possible to excise a carcinoma at the ampulla. In the last two positions it may still be possible to give relief by the performance of a lateral choledochoduodenostomy or by the formation of a new bile duct after the manner used in cases of injury of the duct.

Injuries of the ducts.—These lesions, unfortunately, are not uncommon (Table I). So varied are the anatomical arrangements in this neighbourhood that mistakes at operation are very easy. Not only may the common bile-duct, owing to tension or adhesions, be mistaken for the cystic duct, but it may also be injured in attempts to control bleeding from the stump of the cystic artery which has escaped the ligature. Injuries of this sort are best prevented by strict observation of the rule that in performing a cholecystectomy the cystic duct should never be divided until all three ducts have been exposed and recognized. If the nature of the injury be realized at the time of operation, an immediate end-to-end suture of the ducts will probably give good results. Unfortunately the injury is frequently overlooked, and one of two conditions will then follow. Either a biliary fistula will form and persist, or the wound will heal and a complete obstructive jaundice will develop. In either case the only hope lies in a further operation. In the first variety, with a fistula, difficulties will arise owing to the fact that the bile-duct is small and collapsed and thus difficult to anastomose with the duodenum, but the patient is usually in good condition. When the duct is obstructed the proximal portion is dilated and enlarged and thus can usually be united more readily with the duodenum. The condition of the patient, however, owing to the complete jaundice, will be bad, and especially so if the duct contains white bile.

The technical stages of the operative repair are prolonged and difficult. Much time will probably have to be spent in freeing the structures and in separating adhesions. The distal portion of the duct is usually obliterated and cannot be used for an end-to-end suture. If the proximal portion is sufficiently long it may be directly implanted into the duodenum, but if it is too short for this purpose, a reconstructive operation must be performed.

Carcinoma of the pancreas and chronic pancreatitis.—These two conditions may also give rise to obstructive jaundice. The first will usually be associated with a short history of progressive jaundice and thus will resemble carcinoma of the ampulla. Chronic pancreatitis on the other hand will often present a longer history with previous gastric symptoms and perhaps also one or two attacks of previous transient jaundice. In either case, attacks of severe pain closely resembling biliary colic will be present. In the former, considerable relief, and in the latter a cure, may be brought about by anastomosing the distended gall-bladder to the gastro-intestinal tract. The junction should never be made to the colon, owing to the severe risk of cholangitis, nor to a loop of small intestine, as the diversion of the alkaline bile and pancreatic juice from the duodenum may lead to the later formation—as in one of my cases—of a duodenal ulcer. The operation of choice is the uniting of the fundus of the gall-bladder to the first part of the duodenum, or—if this latter viscus be not sufficiently mobile—to the pyloric portion of the stomach.

Two Cases of Spastic Distension of the Biliary Tract.—CHARLES NEWMAN, M.D.

(I) Mrs. Y., aged 38, suffered from attacks of pain in the gall-bladder region, radiating across the abdomen, and to the angle of the right scapula; these were brought on by exertion or cold weather. Appetite fair; bowels open more than once a day;

respiratory sinus arrhythmia. Gastric residue: Hyperchlorhydria. Duodenal intubation: Delayed response of the gall-bladder to olive oil. Cholecystogram: Good filling, defective emptying. Symptoms relieved very rapidly by prescribed diet and administration of belladonna. Occasional relapses easily cured.

(II) Mrs. M. suffered from attacks of severe pain in the gall-bladder region, radiating across the epigastrium. Much nausea, but no vomiting. Little flatulence. Constipation or lienteric diarrhœa. Appetite poor. Attacks brought on by exhaustion or by being out in the rain. Diagnosis clinical, but condition easily relieved by taking belladonna. Usual subsequent history of occasional relapses, relieved by a dose of medicine.

Valve-stone in the Neck of the Gall-bladder.—CHARLES NEWMAN, M.D.

Mrs. W., aged 52, used to have pain in the epigastrium and scapular region—relieved by vomiting—and attacks of pain in the night, not definitely related to the taking of food. No flatulence or jaundice. A skiagram showed a round shadow in the gall-bladder, representing a combination stone—an original cholesterol solitaire with a secondary inflammatory deposit round it. Gastric residue: achlorhydric. Duodenal tube: Liver bile obtained easily; olive oil produced increased flow of liver bile, but no gall-bladder bile obtained (evidence of the valve action of the stone.) Patient did well on medical treatment for a time, but subsequently had an attack of cholecystitis, on account of which the gall-bladder was removed. The presence of the stone was verified.

Specimen of Gall-bladder with Gall-stones forming beneath the Mucous Membrane.—R. RUTHERFORD, F.R.C.S.

Patient, female, aged 53.

History.—Indigestion eighteen months. No vomiting; occasional jaundice; tenderness in right hypochondrium.

Operation.—The gall-bladder contained many small faceted stones which looked like pigment stones but which when cut were seen to have a cholesterol centre. On examining the mucosa, several small stones were seen to be embedded beneath it; one or two of these stones were actually protruding through, as if on their way out.

The microscopic section shows a mass of mucoid material in a distended canaliculus of the mucous membrane; the whole of the space is not occupied by mucus, there being an empty area from which cholesterol has presumably been dissolved in the preparation of the section.

Comment.—The specimen shows clearly one method, at any rate, by which gall-stones are formed, namely, by a cholesterol nucleus in the depths of a cleft in the mucous membrane. The mucus is most likely an irritation reaction, and when the mass reaches a certain bulk it erupts or extrudes itself into the gall-bladder cavity.

Calcinosis Universalis: A further note on Dr. J. H. Sheldon's Case previously shown.[1]—E. WATCHORN and R. A. MCCANCE.

The example of this rare disease shown at the meeting of the Section held on February 9, 1934, presented one very unusual feature. Some of the calcified plaques were surrounded by fluid and it was suggested that the cavities containing the fluid were enlarged lymph sacs.

A sample of the milky fluid which was first withdrawn by Dr. Sheldon was sent to one of us for analysis and a brief synopsis of the findings may be found in Dr.

[1] *Proceedings*, 1934, xxvii, 623 (Clin. Sect., 45).

Sheldon's article. Later the cavity refilled relatively rapidly and was re-emptied by Dr. Sheldon, who again sent the contents to us. This second sample of fluid did not appear so milky as the first, and its composition was different. Fluid 1 contained calcium and phosphorus in the ratio in which they are found in bone. A number of analyses were made, and although the absolute values differed somewhat owing, probably, to sedimentation of the semi-colloidal suspension and consequent faulty sampling, the Ca/P ratios were constant. Fluid 2 contained approximately the same amount of phosphorus as Fluid 1, but there was a much smaller concentration of calcium. The fluid formed quickly and it seems probable that had it not been withdrawn the calcium would have continued to increase in it till the characteristic bone ratio was reached, for one would expect a static collection of fluid rich in phosphorus to become calcified. In both fluids only a very small percentage of the calcium or of the phosphorus was ultrafilterable.

We make the tentative suggestion that the real disorder in these cases may be the formation of an extracellular collection of fluid rich in phosphorus, and that the calcium is a secondary deposit. In other words, should one regard the disease as a phosphatosis rather than a calcinosis ?

The analytical results of the two fluids were as follows :—

	1	*2*
Ca	580 mgm. per 100 c.c.	123 mgm. per 100 c.c.
P	260 ,, ,, ,, ,,	227 ,, ,, ,, ,,
Ultrafilterable Ca ...	11 ,, ,, ,, ,,	6 ,, ,, ,, ,,
,, P ...	4 ,, ,, ,, ,,	2 ,, ,, ,, ,,
Ca/P ratio	2·22	0·54

Section of Medicine

President—Sir FARQUHAR BUZZARD, Bart., K.C.V.O., M.D.

[*May* 22, 1934]

The Functional Pathology of Advanced Chronic Nephritis

By F. H. SMIRK, M.D.

In advanced chronic nephritis the kidneys have a diminished capacity to form a normally high concentration of urea, creatinine, or sulphate in the urine, and the composition of the urine—as judged by the percentages of urea, creatinine, and sulphate—approaches that of the plasma from which it was formed.

Ordinarily the percentage concentration of every constituent in urine differs from its concentration in plasma, and a change in the concentration of any substance as it passes from the blood to the urine necessitates the performance of work, whether the change is one of dilution or of concentration.

Normally the urine chloride percentage may be greater or less than the plasma chloride percentage and depends on the amount of chloride in the diet. But in contrast to urea, creatinine, and sulphate, the urine chloride percentage in cases of advanced chronic nephritis is almost invariably well below the plasma chloride percentage, even when there is an excess of salt in the diet. Again, in contrast to urea, creatinine, and sulphate, the urine chloride shows no tendency to approach the plasma chloride even in the last stages of renal failure.

On this evidence it is agreed by physical chemists that although the most severely damaged kidneys perform less than the normal work in respect of the urea, creatinine, and sulphate, they must perform more work than most normal kidneys in respect of the chlorides. This capacity to perform work in respect of the chlorides, though apparently unnecessary to the organism, is maintained until the end.

As Mayrs pointed out in 1925, this conclusion seems inevitable physico-chemically but does not make sense biologically. The observations I will describe, together with a number of well-known features of the disease, seem to offer a simple explanation of the difficulty and to reconcile the physico-chemical and biological aspects of the problem.

The experiments were simple. 15 grm. of urea dissolved in 100 c.c. of water were given under comparable conditions to all subjects. A sample of urine was passed before giving the urea and four or five samples were passed at hourly intervals after giving urea.

It was found that in normal subjects urea caused an increase in the urinary chloride excretion and that in patients with nephritis the degree of the increase was less. Also, under the stimulus of urea the percentage concentration of chloride in the urine of normal subjects usually attained a level of 0·8% sodium chloride (figs. 1 and 2) which is above the percentage of chloride in plasma. In patients with advanced chronic nephritis the maximum chloride percentage seldom attained 0·4 % sodium chloride, and in my experience was always less than the chloride percentage in the plasma (figs. 3 and 4).

By determining the plasma chloride, and also in other ways it was shown that the defective chloride excretion in nephritis could not be attributed solely to a depletion of the chloride reserves in the body, and it seemed almost certain that it was mainly due to a defect, of renal—not of extrarenal—origin.

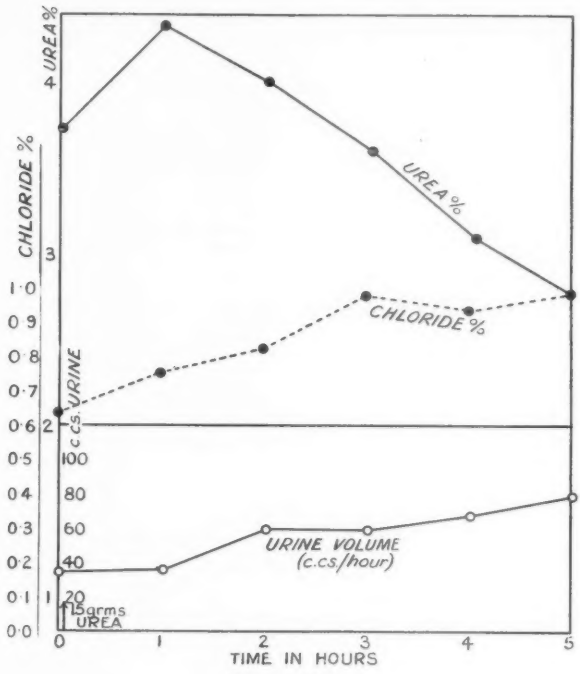

FIG. 1.—Normal.

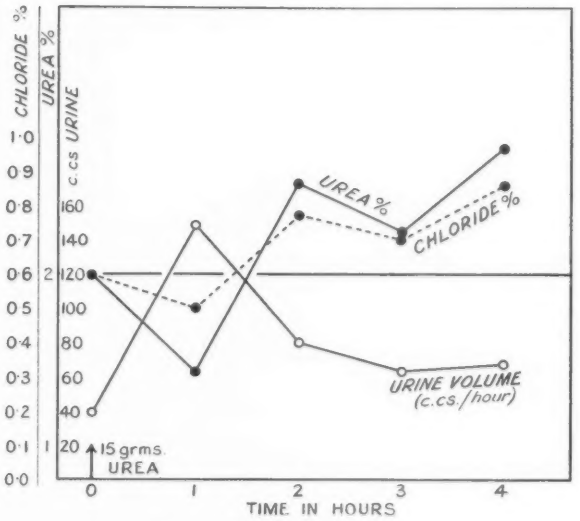

FIG. 2.—Normal.

Also the kidneys of normal subjects were able to increase simultaneously the percentages of urea and chloride in the urine, whereas in nephritis we observed the fundamental difference that a rise in the percentage of urea was almost always accompanied by a fall in the percentage of chloride in the urine (fig. 3). Fixation of the urea percentage was associated with fixation of the chloride percentage (fig. 4).

Now an increase in the urea percentage, by itself, would cause an increase in the total osmotic pressure of the urine, and a decrease in the chloride percentage would cause a decrease in the total osmotic pressure. Often the degrees of these changes

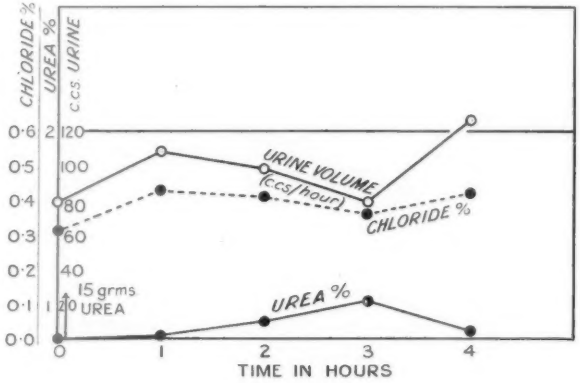

FIG. 3.—Nephritis.

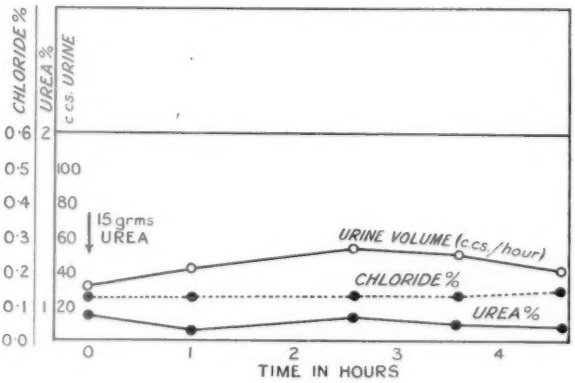

FIG. 4.—Nephritis.

in the percentages of urea and chloride after giving urea are such that they compensate for each other, so that the osmotic pressure of the urine due to urea and chloride is relatively unchanged, but nevertheless, physico-chemically, the fall in the chloride percentage in urine below that in plasma would appear to involve the expenditure of additional energy by the kidney. This difficulty appears to be surmounted in the following hypothesis.

We know already that somewhere inside the renal tubules, even in terminal nephritis, the urea is concentrated locally by the activity of the remaining relatively

healthy cells to above the percentage of urea in the plasma. For this hypothesis, it is of no account whether this concentration takes place by the reabsorption of fluid from the glomerular filtrate or by the secretion of urea into it.

The increase in the concentration of urea will inevitably increase the crystalloidal osmotic pressure of the tubular urine. The suggested hypothesis is that in damaged kidneys some of the renal tubule cells are no longer able to resist the diffusion of water in whatever direction would be determined by osmotic forces. If we accept this hypothesis we must expect diffusion of water from the blood into the tubular urine, for this tubular urine, owing to the concentration of urea, has a greater crystalloidal osmotic pressure than blood.

With sufficient damage of this type, the diffusion of water may continue until the crystalloid osmotic pressures of blood and tubular urine are almost equal—as in fact they may be in advanced chronic nephritis.

In this way it seems possible to explain several features of the disease. The hypothesis accounts, at least in part, for the polyuria of advanced nephritis, because water, diffusing into the renal tubules and rediluting the urea, will increase the volume of urine excreted.

Diffusion of water will also tend to reduce the urinary percentages of substances such as urea, creatinine, and sulphate, which are normally concentrated by the kidneys, and it will cause the percentages of these substances in urine to approach their percentages in plasma.

The low chloride percentage in advanced chronic nephritis, which is well below the percentage of chloride in the plasma, may be explained also by the dilution of the tubular urine with water which diffuses into it through damaged cells. It is because the urinary chloride is not much greater than the plasma chloride, even normally, that on dilution its percentage falls below the plasma chloride.

The inverse relationship between the percentages of urea and chloride in the urine in advanced cases of nephritis is also explained readily by this hypothesis, for, as already indicated, diffusion of water will tend only to equalize the osmotic pressures of urine and plasma, and therefore an increase or decrease in the concentration of one of these constituents (by increasing or decreasing the inward diffusion of water) will cause a relative decrease or increase in the concentration of the others.

Inability to resist the diffusion of water will also explain the inability to dilute much further the already dilute urine of advanced nephritis and to develop a diuresis by water drinking; for diffusion of water back through the damaged cells into the blood would tend to limit the degree of hypotonicity and the volume of urine excreted.

Now posterior pituitary hormone will prevent water diuresis and will control the polyuria of diabetes insipidus but it will not control the polyuria of advanced nephritis. This we can understand very well, if the polyuria of nephritis is due largely to the osmotic diffusion of water into the renal tubules.

The approximation of the reaction of the urine to that of plasma is also explained by dilution of any excess of acid or alkali in the urine by the diffusion of water into the tubules.

By an extension of the hypothesis it is possible to explain the anuria of acute nephritis for, if severely damaged, the cells may become permeable to dissolved constituents as well as to water. In such a case the protein osmotic pressure of the plasma in the tubular capillaries would draw back the fluid previously filtered from the plasma in the glomeruli. That this may actually occur in the frog has been shown in the classical experiments of Richards.

It has been observed recently that although the urinary chloride percentage is low in advanced nephritis, a chloride-free diet is no longer able to render the urine chloride-free. Now according to the present hypothesis, it is a feature of the disease that the efforts of the kidney to perform its normal function of concentrating urea

are frustrated by the diffusion of water back from the blood through the damaged cells into the tubular urine, thus dissipating the potential energy of the urea. The low urine chloride percentage on an ordinary diet represents, in these patients, the dissipation, by water diffusion, of work done previously on the urea in increasing its concentration, and does not represent an active and unnecessary expenditure of energy in depressing the percentage of chloride in urine below that in plasma. But a *further* depression of the chloride percentage in urine below this low percentage reached by passive diffusion would involve the expenditure of additional energy by the damaged kidney. The incapacity of the severely damaged kidney actively to reduce the percentage of chloride in the urine is revealed in the fact that with a chloride-free diet the urine is no longer rendered chloride-free, as in health.

The idea that diffusion either of water or of crystalloids may take place through damaged cells is not entirely new but, so far as I am aware, the consequences of this conception have not been entered into previously in detail. Volhard in his recent monograph quotes two experiments in which the administration of urea depressed the percentage of chloride and the administration of chloride depressed the percentage of urea in the urine of advanced chronic nephritis. He also deduced that the walls of the renal tubules were behaving like semi-permeable membranes, and he related this to the flattening of the renal tubule cells which is a feature of the disorder.

Shaw Dunn (1925) made the suggestion that the damaged renal cells in experimental oxalate nephritis are unable to prevent the indiscriminate leakage of glomerular filtrate with urea into the connective tissue and vessels of the kidney. While this explains the functional pathology of experimental oxalate nephritis, it is not a sufficient explanation by itself for some of the phenomena encountered in human nephritis, as for example the inverse relationship between the percentages of chloride and urea in the urine, and the maintenance of a chloride percentage in the urine below the chloride percentage in the plasma (which is not a feature of the experimental nephritis described by Shaw Dunn).

The hypothesis that some of the damaged cells are unable to prevent the diffusion of water appears to afford a satisfactory explanation of the main relevant features of the disease, but in advancing this view certain reservations must be made. The urine is derived from a large number of units, and in any kidney the degree to which these individual units are damaged, and even the character of the damage, must vary. The urine in advanced nephritis must be a mixture of fluids of widely different compositions, some almost normal, some which show effects of the diffusion of water and some which show the changes described by Shaw Dunn in experimental oxalate nephritis, which probably indicate that the renal tubules are so severely damaged that there is diffusion of urea and chloride as well as of water. Finally there may be a large proportion of renal tissue which is entirely functionless.

It seems unlikely that the functional pathology of advanced chronic nephritis can be explained in terms of a single pathological process, but our present knowledge is in keeping with the view that impairment of the capacity of the renal tubule cells to resist the diffusion of water is a predominant feature of the disease.

A New View of the Intrathoracic and Intrapericardial Pressures in Man

By A. KENDREW, D.M.

In 1815 Dr. James Carson [1], of Liverpool, published a book entitled "An inquiry into the causes of the motion of the blood," and therein set forth his experiments and his views on what is now universally known as the negative thoracic pressure. At that time there was much discussion on the way in which the venous blood was returned to the heart, and supplementary mechanisms for

effecting this return were not infrequently being suggested; Carson's was one of them, and there has been a great deal of subsequent discussion and discovery of how it takes place in the body. There is little to add to-day to what Carson wrote on the elastic traction of the lungs. To the tracheæ of sheep and oxen he connected a glass tube which opened under water, and he observed the issue of bubbles from the tube when the lungs were collapsed by puncturing the thoracic walls. The heart and great vessels, being situated in the thoracic cavity, were, according to him, subjected to this traction and distended, thereby aspirating the venous blood from the systemic veins.

Donders [2] discovered the negative intrathoracic pressure, in man, nearly forty years later, in 1853, and measured it by essentially the same method as Carson. His view of the importance of this pressure for the circulation of the blood may be expressed in his own words:—

"Even during expiration the heart and the vessels in the thorax are under a lower pressure than the other vessels of the body; the venous blood is therefore continuously aspirated towards the thoracic cavity."

Donders appears not to have heard of Carson's work.

This idea of an aspiration effect produced by an intrathoracic negative pressure had not been called in question when I drew attention in 1926 [3] to the discrepancy existing between current opinion and the actual facts of hydrostatics.

For many years the intrapleural pressure has been measured in man by connecting a U-tube manometer to a hollow needle inserted into a pleural cavity.

In 1919 Morriston Davies [4] stated that:—

"The intrapleural negative pressure is equal, in a normal subject, to about 6 mm. Hg, and shows only a slight excursion with inspiration and expiration. The excursion may be as much as 1 mm. Hg during quiet respiration, but is as a rule less. . . . With deep inspiration or forced expiration the negative pressure may be increased or diminished a further 1 mm. Hg."

About twenty years ago some physiological work was done which seemed to support Carson's theory. Knowlton and Starling [5] showed with the heart-lung preparation that the diastolic filling of the heart largely conditioned its output, after Lewis [6] in 1908 had proved how great was the influence of a small positive pressure in the pericardial sac in causing a fall of arterial pressure. Lewis was of the opinion that the intrapericardial pressure had never been satisfactorily measured in animals; it was, he wrote, usually regarded as the same as the intrapleural pressure.

In man the intrapericardial pressure has never been measured. This omission is all the more remarkable in that, although the general physiological influence of the negative intrathoracic pressure on the circulation has been described in all the recent textbooks, the pathological alterations of the pressure conditions have remained largely unknown, in spite of the widespread use of artificial pneumothorax in the treatment of pulmonary lesions of many kinds.

It is desirable to digress for a moment and stress the importance for our argument of obtaining a clear idea of what is entailed in the experimental work on the intrapericardial pressure in animals; the pressure is raised by running saline into the pericardial sac and thus necessarily diminishing the volume of the heart in diastole. The conditions are artificial and the conclusions, however interesting, have no direct reference to the intact animal.

Shortly after the Great War, interest in the venous pressure in man in normal and pathological conditions began to be manifest in a large number of clinics in Europe and America, and measurements of the pressure in the systemic veins were made in several ways and yielded results which were subjected to a rather premature classification and much immature deduction. The aim of the bulk of this work was to

estimate the pressure in the right auricle and the general venous cistern, with the subject in the recumbent position, and the reasonable assumption was made that the systemic veins approximated very closely to a hydrostatic system. After making a large number of peripheral measurements, it became abundantly clear to me that their reference to central conditions, to a right auricle which lay securely in the middle of what we had been accustomed to regard as a zone of negative pressure, was unjustified without a knowledge of the degree of this negative pressure, which might vary within unknown limits, at least in pathological subjects. In other words, the venous channel from the veins of the forearm to the right auricle was not a simple system of tubes subject to the laws of hydrostatics, and could not be used as a manometer. The more one attempted to analyse the system as it was then described, the more bewildering became the gradual, or, it might be, sudden change in external pressure alleged to be suffered by the veins of the body as they passed behind the sternum and approached the heart; was there a point where the pressure became negative? The fallacy lies, however, not with the method of determining venous pressure, but with our past failure to see that the chest consisted of two parts—one, bilateral, enclosed by the parietal pleuræ, and the other the mediastinum; in the intrapleural spaces a measured negative pressure prevailed, but in the mediastinum no mechanism had been provided to alter the atmospheric pressure which is found throughout the body. A schematic description of the human thorax will make this quite clear.

The thorax is a barrel-shaped cavity which tapers towards the upper end, and is there closed by the fasciæ covering the domes of the pleuræ, and by the large vessels and other structures which pass embedded in fascia to enter the thoracic cavity. The lower end of the thorax is closed by the diaphragm, which consists of two parts, a central fixed tendon and a peripheral muscle. On the tendon of the diaphragm is fixed the pericardium, which encloses the heart and a certain portion of the large vessels, passing at the pericardial reflexion to blend with their coats. The pericardium is held in place, then, by the tendon of the diaphragm and by the fasciæ in the upper part of the thorax. The fibrous pericardium is a strong indistensible bag. The central part of the thoracic cavity, the mediastinum, is formed of the somewhat conical tube with firm walls, consisting below of the tough pericardium, and above of the tissue in which the great vessels are fixed. Round this tube lies the second part of the thorax, the two potential spaces enclosed by the parietal pleuræ, which are firmly attached to the bounding walls of the thorax, except where these membranes pass round the pericardium to which they are again attached. In these two potential spaces the lungs are suspended, and expand to fill them, stretching the elastic fibres which are an important part of pulmonary tissue, for the pull of these fibres is responsible for the negative intrapleural pressure, which subsists even at the end of expiration. In short, the lungs are always too small for the space they occupy. The divisions between the two parts of the thorax are strictly demarcated; there is the mediastinum whose structure is not in any way peculiar, and there are the two separate cavities bounded by the parietal pleuræ, furnishing with their contents an organ admirably designed for the purpose of ventilating the lungs. The bellows action of the thorax is efficient only if the boundary common to intrapleural spaces and mediastinum is unyielding.

When a deep inspiration is taken the intrapleural pressure may fall to -7 mm. Hg, and this would be the unsupported pressure exerted by the atmosphere on all the walls bounding the cavity: if these walls are unresisting, they will be drawn into the pleural space till that space is sufficiently diminished for the pressure therein to attain that of the air outside. But this does not happen, for the walls which bound the cavity are the bony and muscular thoracic cage, the muscular part of the diaphragm, and, towards the middle of the thorax, our conical bag of pericardium and fascia. The inferior surface of the central tendon of the diaphragm is unques-

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tionably exposed to the full atmospheric pressure transmitted by the abdominal viscera, and this pressure must be transmitted through the diaphragm to the pericardial fluid and the heart, for there is no device to prevent it.[1] It is equally clear this atmospheric pressure is transmitted through the walls bounding the pleural spaces, but owing to the presence of the lungs with their elastic tissue in tension, a manometer connected to a pleural space will record a negative pressure. I wish to emphasize what I am sure is now obvious, that there is no mystery in the transmission of the atmospheric pressure through the diaphragm to the pericardial and mediastinal contents, and that, moreover, if the elastic tissue of the lungs were entirely transformed into fibrous tissue, the pressure in the pleural cavities would also become atmospheric.

Having disposed of the concept of a negative intrapericardial pressure, it remains to deal with the general physical principles involved in Carson's original theory. He supposed that the mere presence inside the thorax of a negative pressure sufficed to aid the return of venous blood to the heart. The principle of the conservation of energy immediately excludes any possibility of such action, for work must be done when the blood is being returned to the heart against gravity, and none can be furnished by a negative pressure which remains at a constant value.

I hope that this short outline, from a physical point of view, of the pressure conditions in the chest will stimulate investigation of some of the rarer congenital defects of the cœlomic cavity and its boundaries, such as the unilateral communication of pericardial and pleural spaces.

It would be beyond the scope of this paper to do more than mention the bearing it has on many of the methods of estimating the cardiac output, and especially on the theories underlying them.

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5 Knowlton and Starling, *Journal of Physiology*, 1912, xliv, 206.
6 Lewis, *ibid.*, 1908, xxxvii, 212.

Other papers read at this meeting will be published in the next issue of the Proceedings.

1 The atmospheric pressure is transmitted to the upper surface of the pericardium by the structures which pass behind the manubrium sterni into the upper part of the mediastinum.

Section of Urology

President—A. CLIFFORD MORSON, O.B.E., F.R.C.S.

[*May* 24, 1934]

Endometriosis of the Bladder

By Dr. J. A. WEIJTLANDT (Amsterdam)

BY endometriosis we mean an affection occurring in the female genital organs and occasionally in neighbouring parts of the abdomen which anatomically and functionally resemble the mucous membrane of the uterus (endometrium).

We are still ignorant of the way in which, and the reason why, it develops. Many theories have been advanced to explain its origin, but none have proved satisfactory.

Though I do not presume to be able to solve so difficult a problem I think some peculiarities of my own case might throw a little light upon it.

Endometriosis is characterized by ducts, lined with cylindrical epithelium lying in the typical so-called " cytogene " stroma of the uterus mucosa.

We distinguish two groups of cases. In the first there is a direct connexion between the normal endometrium and the dystopic endometrium-like tissue. There is no doubt that here the endometriosis develops from the endometrium by an abnormal propagation into the muscular layer of the uterus. This propagation proceeds by a solution of the fibrous tissue in the muscle; the muscular tissue itself remains unchanged. The second group develops without any direct connexion with the mucosa of the uterus. The growths are found in the ovary, round ligament, pouch of Douglas, rectum, cæcum, ileum, umbilicus, groin, and bladder, and on the serosa of the uterus and fallopian tube.

Endometriosis of the bladder is a very rare affection of which I could find only 23 published cases.

The patient in my case was a woman, aged 31. As a child she had had scarlet fever. She had begun to menstruate at the age of 15; the hæmorrhages were frequent (every two weeks) and of long duration (from six to eleven days). After a year the discharge became still more profuse, now and then causing fainting. During the next four years these symptoms remained, alternating with shorter periods of rather normal menstruation.

In 1923, when aged 19, she was treated by curettage. This effected only a temporary improvement. Menstruation ceased for about a year; then menorrhagia recurred. Another curettage, in 1926, had no influence on the symptoms. In 1927, in another hospital, she was treated by clamps on the parametria through the vagina (Henkel's method), with no better result.

On March 4, 1929, she entered the Burger Ziekenhuis at Amsterdam in a very miserable condition, weak and extremely anæmic. The hæmoglobin index was 18 (Sahli); the urine was normal. 450 c.c. of blood was given intravenously. The next day she looked better; the hæmoglobin index was 32. She continued to improve, and the hæmoglobin index rose to 85. Dr. Slingenberg, the gynæcologist at the hospital, found no abnormality of the genital organs. The menstruation remained somewhat profuse, but not extremely so. The patient was discharged from hospital on May 25, 1929.

During her first months at home, however, menstruation became more and more profuse, and the patient lost so much blood, that she was again admitted to the hospital, on October 24, 1929. The general condition was good, though the hæmoglobin index was only 47. In the course of the following months she was treated with different remedies without any effect on the profuse menorrhagia. At last Dr. Slingenberg performed a laparotomy, which revealed no pathological changes except some small cysts in both ovaries; a cuneiform excision was made from both ovaries. The result was a definite decrease of the menstrual flow, which, however, persisted with irregular intervals. She was dismissed with slight continuous fluxus on June 13, 1930. Yet the end-result of this operation proved to be a failure, for the profuse menorrhagia soon recurred and the patient was admitted to hospital, for the third time, September 6, 1930. Because of the low hæmoglobin index (37), another transfusion was given. On October 24, 1930, Dr. Slingenberg performed a supravaginal hysterectomy and removed the right ovary, which had formed a cyst as large as an orange. After an uneventful recovery, the patient left hospital, November 17, 1930, apparently cured.

The gross anatomical examination of the uterus did not show the cause of the bleeding. This, however, was revealed by the histological examination (Dr. van Hasselt), which showed a typical endometriosis—a so-called internal endometriosis of the uterus.

After the operation menstruation did not recur. Thus it seemed that the rather drastic therapy had finally succeeded in curing this unfortunate young woman.

FIG. 1.—Cystoscopic appearance of endometriosis of the bladder.

To our great disappointment, however, scarcely six weeks after the operation a new symptom developed, namely, frequent and painful micturition, the urine being mixed with blood. This lasted from the beginning of December until Christmas 1930. Then followed a week without any complaints. In the first week of January 1931, the frequent painful passing of urine containing blood recurred and persisted for three weeks. The last week of January was again without trouble. This same alternating course was observed during the following months.

The patient again entered hospital, January 28, 1931. The urine contained albumin and blood-cells.

Cystoscopic examination showed a swelling of the bladder mucosa over and immediately above the inter-ureteral ridge. This swelling reached as far as both ureteral orifices. The left orifice was lying within the swollen region, the right one just at its border. The greater part of the surface of the tumour-like part presented the appearance of so-called "bullous œdema." Near the left ureteral orifice there was a small area with a more tumour-like appearance, in which were two little vesicles with blue centres, looking as if filled with blood (*see* fig. 1).

The dorsal circumference of the bladder-neck was irregular, with gross knobs and bullous œdema, here and there resembling polyps.

At first sight these cystoscopic findings might have suggested the diagnosis of neoplasm, or of an inflammatory process extending into the bladder from without. The previous histological report on the uterus and the resemblance of the bladder condition to the descriptions by Müller, Ottow, and others in similar cases, led to the diagnosis of endometriosis of the bladder. Excision of several portions of the altered mucous membrane of the bladder by means of Young's cystoscopic rongeur did not, however, yield sufficient tissue to confirm this diagnosis histologically. Nevertheless the regularity with which the periods of frequent and painful passing of urine containing blood alternated monthly with periods of freedom lent strong support to it.

Every monthly period consisted of three weeks of bleeding followed by about one week of normal health. During the first three weeks the urine contained albumin and blood; in the last week of each period there was no albumin—or only a trace of it—and there were no red blood-cells (though there were a few white ones). Cystoscopic examination showed an alternating increase and decrease of the swelling of the bladder mucosa in accordance with the clinical symptoms.

As mentioned above, both ureteral orifices were more or less involved in the pathological process. Yet good secretion of indigo-carmine showed that the function was unimpaired.

The localization of the endometriosis in the dorsal part of the bladder implied that—as in most published cases of bladder endometriosis—there had been a direct continuation of the morbid affection from the uterus to the bladder wall. A minute examination of the excised uterus, however, showed, that in this organ the affection had not even reached the outer layers of the muscular coat. Further, during the operation Dr. Slingenberg had not observed anything abnormal in the region between uterus and bladder. (In a later operation for appendicitis this was verified.)

We, therefore, came to the conclusion that we had to deal with endometriosis situated in two different places, independent of each other, the first being the uterus and the second the bladder, or, in the terms of R. Meyer, *adenomyosis uteri interna* and *adenomyosis vesicalis interna*. This last form of endometriosis is very rare. Of the 26 published cases of endometriosis of the bladder only three[1] belong to this variety, which we would prefer to call *primary endometriosis of the bladder*.

The choice of the treatment constituted a further difficult problem.

The open operation—excision of the diseased tissue through a suprapubic cystotomy—appeared likely to be most effective, but the close relationship of the ureters made it doubtful if a radical excision would be possible without necessitating reimplantation of the ureters. Moreover, the patient absolutely refused to undergo an operation.

Electro-coagulation seemed likely to be both insufficient and dangerous, because of the depth to which the pathological processes had penetrated the bladder wall.

Another idea which I entertained was the possibility of influencing the menstrual cycle by changing the hormonal relations in the patient's blood. Professor Laqueur, who advised me on this delicate question, after having daily examined the urine of the patient during the whole of a four weeks' period, could not suggest a rational therapy. Therefore I abandoned that idea also.

The only remaining possibility was to try to eliminate the influence of the remainder of the left ovary on the endometrioid tissue by means of Röntgen castration, and after due consideration this was carried out. It was performed by Dr. Heilbron in four sessions, namely April 22 and 24 (1931) in the short complaintless

[1] Müller, *Archiv. f. klin. Chir.*, 1927, cxlv, 394; Frommolt, *Zentralbl. f. Gynæc.*, 1927, p. 1159; Oehlecker, *Zentralbl. f. Chir.*, 1930, p. 2.

period (intermenstruum), and on May 8 and 11 in the middle of the next hæmorrhagic period. At first the result was not very satisfactory. On October 29 (six months after the first radiation) the subjective symptoms disappeared after having lasted only seven days. The patient remained free from the trouble for forty-three days. Then came a short relapse (December 11 to 20) which was followed by further radiation treatment. After that the patient remained without trouble and without bleeding.

The cystoscopic control had demonstrated that from the month following the X-ray treatment the extent of the endometriosis decreased gradually. On November 27, 1931, there remained but little of the serious changes which the bladder had presented. During the period of relapse in December 1931 the bladder showed this clearly also.

On February 2, 1932, the bladder appeared to be normal; only a slight irregularity at the left dorsal side of the bladder neck had persisted. The urine contained no albumin and only sporadic white blood-cells. Vaginal examination, however, revealed a small, rather soft swelling in the posterior bladder wall.

Unfortunately, this good result did not last longer than three and a half months. Then the attacks of painful passing of blood-tinged urine recommenced (April 27, 1932). The monthly hæmorrhages increased gradually so that in June 1932 there was again a bleeding period of three weeks followed by a free pause of about one week.

A complementary series of Röntgen radiations was unsuccessful. Though her condition was rather deplorable our patient remained at home in the country until an attack of appendicitis brought her again to the hospital (December 1932). Here she was treated by Dr. Knapper who, when performing the appendicectomy, had the opportunity of examining the abdominal cavity, and endeavoured to find the remainder of the left ovary with a view to removing it. It was very difficult to find anything resembling an ovary and the excised part (after histological examination) appeared to contain no ovarian tissue and consisted of fibrous tissue only. As the operation had no effect on the periodical hæmorrhages from the bladder, we concluded that there was still more or less ovarian tissue remaining.

Towards the end of 1933 the periodical hæmorrhages grew more and more serious. The patient returned to the Burger-Ziekenhuis on February 20, 1934, in a wretchedly anæmic condition (hæmoglobin index, 27). A blood-transfusion (450 c.c.) was carried out on the day of admission and improvement followed rapidly. Within two weeks the hæmoglobin index rose to 63 and the number of red blood-cells to 4 million. Cystoscopy, however, still showed the same typical appearances.

What could we do for her now? Röntgen radiation appeared to have failed. The drawbacks to operative treatment were about the same as they had been three years ago.

My opinions as to the necessity of complete removal of the endometrioid tissue had changed in the meantime, after several cases of bladder endometriosis, which had healed after partial excision had come to my notice.[2]

As I considered electro-coagulation to be the simplest method for partial destruction of the endometrioid tissue in the bladder wall, I resolved to try it. Under sacral anæsthesia with 2% novocain, I have now performed it three times at intervals of eight or ten days. There has been no disagreeable reaction, except painful micturition during the first days after the treatment.

Though I cannot yet indicate the final results—one or two more sessions of coagulation treatment will be necessary—I think that at present the effect is quite satisfactory.

[2] Schindler, *Frankf., Zeitschr., f. Path.*, 1925, xxii, 189; Keene, *Ann. Journ. Obst. and Gyn.*, 1925, x, 619; Plaut, *Zentralbl. f. Gyn.*, 1929, p. 3358.

The history of my patient induces me to make some remarks concerning the following three points:—

(1) The theory of the genesis of endometriosis in general; (2) the symptoms and some other peculiarities of endometriosis of the bladder; (3) the treatment of this last affection.

(1) The theory of De Josselin de Jong and De Snoo[3] (that endometriosis originates in special parts of the cœlom, which have preserved their embryonic potency and afterwards develop the properties, belonging normally to the female genital tract) has my support, but it does not explain the cases of autochthonous endometriosis of the bladder wall, having no direct connexion with the peritoneum or the uterus.

Now, in most of the published cases of endometriosis of the bladder, the distal posterior part near the ureteric orifices was affected. It was the same in my own case, but in that case the dorsal part of the bladder neck also showed analogous alterations.

If we assume that endometriosis develops from a special part of the cœlom, it is impossible to explain its presence in the bladder wall independently of the serosa. The cœlom has never had anything to do with the urogenital sinus, a part of which afterwards becomes the bladder.

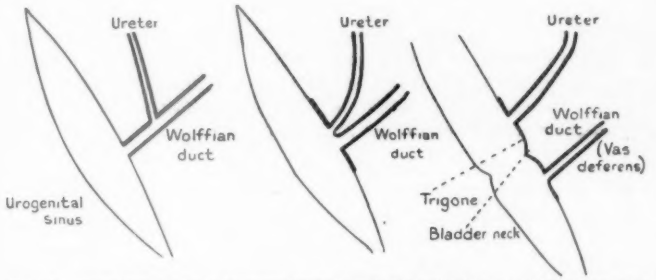

FIG. 2.—Development of the mesodermal part of the bladder. (After Broman.)

This problem may be solved if we assume that not only the cœlom, but also other derivates of the third germ layer, the mesoderm—in the range of a certain number of abdominal segments—can preserve that potentiality of developing into tissue, which ordinarily is the exclusive possession of the Müllerian ducts.

In the bladder it is exactly the region affected by endometriosis (trigone, ureteral orifices, dorsal part of the bladder neck) which develops from the Wolffian ducts and thus is of mesodermal origin, whereas the remainder is endodermal. (*See* fig. 2, after Broman.)

Thus, actually, the cases of autochthonous endometriosis of the bladder support, rather than oppose, the theory of De Josselin de Jong and De Snoo.

(2) Endometriosis of the bladder is a rare disease, at any rate only a small number of cases has been published. In England, Clifford Morson published a case in the *British Journal of Surgery*, 1927, and Ottow (Berlin) gives in the *Zentralblatt für Gynäkologie*, 1929, p. 3330, a concise synopsis of 16 published cases, of which two are his own. In the later literature I could collect only six other cases.[4] In most of these cases the endometriosis had reached the bladder from the outside,

[3] De Josselin de Jong and De Snoo, *Virchow's Arch.*, 1925, cclvii, 23.

[4] Joseph, *Z. f. Urol. Chir.*, 1930, xxix, 396; Cilento, *Journ. d'Urol.*, 1932, xxxiv, 247; Frommolt, *Zentralbl. f. Gyn.*, 1932, 1707; Eberhard, *Zentralbl. f. Gyn.*, 1932, 1712; von Sauer, *Zentralbl. f Gyn.*, 1933, 347; Settergren, *Acta, chir. Scandin.*, 1933, lxxiii, 312.

from the excavatio vesico-uterina, only in three cases it appeared to have developed in the bladder wall itself (*see* p. 53).

As in most of the published cases the diagnosis has been made after operation and histological examination, it will be useful to indicate the symptoms which may enable us to recognize this affection by clinical and cystoscopic examination.

(*a*) Endometriosis of the bladder can be diagnosed only during the period of menstrual activity, not before the beginning of menstruation and not after the menopause.

(*b*) The disturbances are periodical, coinciding with the menstrual cycle.

(*c*) In the menstrual period there is frequent painful micturition, and the urine contains albumin and blood. In the intermenstruum there are no, or only slight, subjective complaints.

(*d*) Bimanual vaginal examination may detect a soft swelling, which is tender during menstruation.

(*e*) Cystoscopy reveals the true nature of the affection. There is swelling of part of the mucous membrane of the posterior wall of the bladder. This locality is explained by the position of the uterus in the case of endometriosis from that organ, and by the mesodermal origin of the trigone, ureteric orifices, etc., in the case of autochthonous endometriosis of the bladder.

(3) The treatment of endometriosis of the bladder differs according to the age of the patient. In women who are near the menopause, castration by means of Röntgen-radiation is the simplest method. In younger women, the most efficient treatment is radical excision. This should always be performed by laparotomy, in order that one may be informed of the relations of the bladder endometriosis to the internal sexual organs and the peritoneum. If this operation, for any reason, is not feasible, intravesical electro-coagulation may be tried.

Section of Obstetrics and Gynæcology

President—W. BLAIR-BELL, F.C.O.G.

[*June* 22, 1934]

A Case of Conjoined Twins.—DAVID CURRIE, F.R.C.S.

The mother, a primigravida, was admitted into the hospital on November 28, 1933, complaining of acute abdominal pain. Her last menstrual period had been on May 23, and the confinement was expected on March 2, 1934. Previous to the onset of pregnancy she had had few illnesses until two years ago when she was under medical care on account of anæmia. Menstruation had begun at the age of 16 and had been normal. The history of the pregnancy was uneventful until October when she noticed that her body was rapidly increasing in size. This increase progressed steadily, but caused little discomfort until November 27, when she complained of abdominal pain. Her doctor saw her, and requested her admission to the hospital.

On admission, the temperature was 97·2, and the pulse-rate 96. There was no albumin in the urine. The abdomen was distended by a large uterine swelling, which reached up to the xiphisternum. The umbilicus was flattened and there were numerous distended subcutaneous veins. The uterus was not tender. No fœtal parts could be felt and no fœtal heart could be heard. A vaginal examination revealed the presence of a fully formed cervix, but again, no fœtal part could be felt. The condition was diagnosed as hydramnios, of the subacute type, causing abdominal discomfort, and in accordance with the custom of the hospital, an X-ray photograph was obtained. This showed the presence of an unusual fœtus. A single head could be identified lying at the brim of the pelvis, and above it were two bodies. The two spines converged and appeared to unite at the base of the skull. Because of this abnormality it was decided that the pregnancy should be terminated. The fœtal head, though larger than that usually associated with a twenty-eight weeks' fœtus, was considered to be small enough to enter the pelvis. Labour was induced by puncture of the membranes at 10.30 p.m. on November 30, and 10 pints of liquor amnii were allowed to escape; a normal delivery occurred twenty-seven hours later. After the membranes had been ruptured a fœtal heart could be heard, but the monster was born dead.

The fœtus was lying in a single sac of membranes; there was only one placenta, and one cord, although the latter could be divided into two separate components twisted around one another.

The fœtus belongs to the type of Janus monster classified as "syncephalus-monoprosopus," one of the commoner types of conjoined twins. The twins are joined together at the head and thorax and separate below the umbilicus. There is one face with duplication of the occipital regions. The bodies are joined together front to front, the faces are turned laterally and the composite face is made up of the right side of one face and the left of the other. This fœtus has not been dissected, but in most cases of this type, the brain shows evidence of duplication, especially in the region of the medulla, cerebellum, and pons.

Because of the large size of the composite head, a craniotomy is usually necessary if such a fœtus is carried to full term, and the value of a routine X-ray examination, in cases of hydramnios, is clearly shown in this case. During the last two years such a procedure has been the custom of the hospital, and we have been surprised to find what a large number of anencephalic monsters can be diagnosed, previous to the onset of labour. Thus, in 22 such examinations an abnormal fœtus was discovered on 13 occasions. In 11 cases the malformation was one of

anencephaly, in one of hydrocephaly and in another of syncephaly. The clearest pictures are obtained when the mother is photographed while lying on her side.

In all instances of gross fœtal malformation, labour was induced by puncture of the membranes. This was carried out by an instrument especially designed for the purpose, consisting of a long catheter with both a terminal and a lateral eye. Through the terminal opening a stilette can be made to protrude a quarter of an inch. When the stilette is withdrawn into the catheter, the latter can be passed with little danger through an undilated cervix and then the membranes punctured according to the method described by Gibbon Fitzgibbon. The liquor in cases of hydramnios runs away in a steady yet slow stream. The value of the instrument was demonstrated in the case of the hydrocephalic fœtus. In this a large head was found during the antenatal supervision, and proved to be hydrocephalic by an X-ray examination. The membranes were ruptured through an undilated cervix, and then the point of the catheter was carried through a fontanelle into the skull, liberating 600 c.c. of cerebrospinal fluid. By this means the head was reduced to a small size, and labour was induced at the same operation. Labour progressed satisfactorily and was completed within twenty-four hours.

At the Leeds Maternity Hospital labour has been induced by puncture of the membranes 230 times during the last two and a half years, the principal indications being slight degree of contracted pelvis, albuminuria and pre-eclampsia. Because of the excellent results obtained in pre-eclampsia, labour has been induced in cases of eclampsia during the last year by the same method.

The following figures may be of interest. The average time of labour in all primigravida (including cases of contracted pelvis) was twenty-five hours, and in multigravida fifteen hours. When induction of labour was undertaken on account of contracted pelvis the times were longer, namely thirty-two hours forty minutes, and thirty-one hours fifty minutes respectively. The morbidity rate was 4·3% and the maternal mortality rate 0·43% (this was a patient with severe accidental hæmorrhage). If cases of anencephaly, antepartum hæmorrhage, death of the fœtus in utero, and eclampsia are excluded, 26 children were either born dead or died shortly after birth, mainly from prematurity—a fœtal mortality rate of 11·2 %.

Discussion.—The PRESIDENT said that he had been glad to hear Mr. Currie speak of the routine X-ray examination of pregnant patients, which had led to such an excellent diagnosis and to the proper treatment in his case. He (the President) had for many years had a routine radiographical examination made in all cases of suspected structural anomalies of the fœtus or the mother. In this connexion he and his colleagues, Mr. Thurstan Holland and Dr. R. E. Roberts, had been much interested in the question of maturity, as shown by centres of ossification, and of postmaturity.

Mr. L. C. RIVETT asked Mr. Currie how he had tapped the hydramniotic sac in the case of twins with anencephaly and hydramnios in the upper sac.

Mr. CURRIE (in reply) said that the normal fœtus was lying in the lowermost sac.

A Case of Chorion Epithelioma of the Uterus confined entirely to the Myometrium, diagnosed by the Zondek-Aschheim Test.—JOHN BEATTIE, F.R.C.S.

The uterus and both appendages were removed from a woman aged 33. The uterus is normal in size ; the endometrium is smooth throughout and normal in appearance. In the myometrium of the left cornu of the uterus is an area of chorion epithelioma, half an inch in diameter, which has no connexion with the endometrial lining of the cavity of the uterus. Both ovaries have been removed. One contains a unilocular cyst one inch and a half in diameter ; the other is enlarged to a small degree and contains a large number of areas of granulosa lutein tissue which can be seen macroscopically.

The case history is as follows :—

In 1933, after seven weeks' amenorrhœa, the patient suffered from irregular bleeding for four months and finally delivered herself spontaneously of a vesicular mole. Convalescence was uneventful and menstruation recommenced seven weeks later with a cycle of fourteen to twenty-eight days, whereas previously the cycle was regular at twenty-nine days. The menstrual loss at this time was normal, but occasionally between her periods she suffered from a thin, blood-stained discharge. The last regular period was January 6 to 10, 1934, and two weeks later she began to bleed irregularly and continuously.

On vaginal examination the uterus was found to be not palpably enlarged and the cyst in the right ovary was easily felt. At this time the uterus was curetted with a sharp instrument; there was no abnormal bleeding and no evidence of chorion epithelioma was found in the material removed.

The Zondek-Aschheim test was performed on several occasions as follows: Five infantile female mice were used on each occasion and each one received six injections of 0·4 c.c. of urine subcutaneously. The urine used was always a morning specimen. The mice were killed ninety-six hours after the first injection.

The first test was normal positive.

The second test was normal positive with dilution of urine to one in twenty, but negative with dilution of one in one hundred.

The third test was only very weakly positive with normal undiluted urine and negative with dilutions of one in twenty. This was carried out one week after the second test.

The fourth test was carried out fourteen days after the third test and was normally positive with undiluted urine.

At this stage panhysterectomy was performed and the convalescence was uneventful.

Nine days after operation the Zondek-Aschheim test was negative with normal undiluted urine and has remained negative until the present time, i.e. six months. The test has been made monthly since operation.

Histologically the specimen is of interest, in that the cyst in the right ovary is lined by granulosa lutein cells and the left ovary contains several areas of hyperluteinization which, by serial section, are shown to have no direct connexion with each other. These areas have a structure very like the corpus luteum and suggest the possibility of multiple ovulation with rapid luteinization of the resulting collapsed follicles. In other parts of the ovary are several cystic follicles which are not luteinized. There are no metastases in either ovary and the structure of the growth in the myometrium of the uterus is typical of chorion epithelioma.

The endometrium contains glands and stroma in the resting stage of the menstrual cycle and there are a few glands present with cystic dilatation in some areas.

Thanks are due to Dr. Brewer of St. Bartholomew's Hospital for the help given in performing the numerous Zondek-Aschheim tests in this case.

The PRESIDENT said that Mr. Beattie's case was remarkable in the variation that was found in respect of the Zondek-Aschheim reaction: if the growth was not disturbed by the curette there must surely have been some error of technique in the third test.

The Zondek-Aschheim Test after Partial Hypophysectomy

By D. M. STERN, F.R.C.S.

THE patient, an unmarried woman aged 21, was admitted to the London Hospital under the care of Mr. Hugh Cairns in July 1931.

There was a month's history of visual disturbance, but fifteen months previously she had noticed a progressive enlargement of her hands and feet. On examination

she was slightly acromegalic, with bi-temporal hemianopia of scotomatous type. A skiagram showed enlargement of the sella turcica. That the acromegaly was not marked is obvious, for she was engaged to be married.

She had first menstruated at the age of 14, and her periods, which never became regular, with intervals of from six weeks to three months, lasted about six days. Two and a half years before her admission to hospital they had ceased altogether, except for one period, four months previously.

A few days after admission, Mr. Hugh Cairns removed most of a cystic tumour of the anterior pituitary through a right trans-frontal flap. Dr. Dorothy Russell reported on the tumour as follows:—

"Fragments of solid, polygonal-celled, predominantly alpha-granuled adenoma of the pituitary. The largest fragment is a portion of anterior lobe. Others mainly composed of stout collagen-containing fibres of (?) remnants of anterior lobe. In addition, there are a few fragments composed of small polygonal cells separated by numerous red corpuscles and neutrophilic leucocytes. These cells mostly contain alpha granules."

The patient made a rapid recovery from the operation and from the bi-temporal hemianopia, and the acromegalic appearances became less pronounced. In August 1931 she married, six weeks after the operation. She never menstruated again, but one year later (in September 1932) she noticed that her abdomen was swelling and she felt movements. On examination she was found to be five months pregnant. She had a normal pregnancy, and was delivered in January 1933 of a live girl weighing 7½ lb. after a normal labour lasting twenty-one hours. Six weeks after delivery she had some uterine bleeding for three days, but she has had no menstrual period since then.

She says that, so far as she knows, her sexual feelings are normal, nor did she notice any change in her libido after the operation. No contraceptives have been used.

The Zondek-Aschheim reaction at the twenty-fifth week of pregnancy was positive, though the concentration of gonadotropic hormones was definitely below normal. Six weeks after delivery the reaction was negative.

Conclusions.—Normal pregnancy and labour occurred in a patient who was not menstruating, and in whom there can have been very little anterior pituitary substance. Presumably the secretion from the anterior pituitary was insufficient to stimulate the ovary to normal regular ripening of follicles with production of corpora lutea. The Zondek-Aschheim reaction was positive, in spite of the deficiency in the pituitary. Are we justified in assuming that the hormone found in the urine has some other source—such as embryonic trophoblast?

My thanks are due to Mr. Cairns and to Mr. Victor Lack for permission to discuss this case, and to Dr. Russell for the pathological report.

Discussion.—The PRESIDENT said that Mr. Stern's case was of interest in regard both to the pathology of the pituitary lesion and to its effects on the genital functions. A cyst of the pituitary was usually associated with dystrophia adiposogenitalis—owing to pressure atrophy of the cells of the pars anterior—and not with acromegaly. He would like to know whether glycosuria was absent in this case and, if so, what was the result of the sugar tolerance test. He doubted whether the successful operation was responsible for the ovulation and pregnancy which occurred in the absence of menstruation—not an uncommon occurrence—for the patient might have been ovulating previously: she had married after the operation. That the Zondek-Aschheim reaction was positive meant little, as living syncytium was present, and in any case the removal of the pars anterior had obviously been partial only, otherwise ovulation could not have taken place.

Mr. GREEN-ARMYTAGE said that at the Congress of Gynæcologists in Glasgow, he had shown a complete series of skiagrams in a case of acromegaly during pregnancy.[1] The

[1] This case was recorded, with a photograph, in the *Clinical Journal*, 1931, lx, 274.

signs and symptoms in this case had developed acutely at about the third month of pregnancy, and had gradually become worse with marked ocular and mental indications. After confinement, which went to term, the patient became quite normal mentally. Eighteen months later, after complete amenorrhœa, she again became pregnant and went through the same cycle of acromegalic symptoms. Obviously this was a case of anovular menstruation with pregnancy. An interesting fact, but perhaps one to be expected in this case, was that during the carrying period and after parturition there was no mammary secretion, the breasts being small and dry. What had Mr. Stern's findings been in this respect?

Mr. STERN (in reply) said that there had been no glycosuria and the fasting blood-sugar before operation was 0·08%. The baby had been breast-fed. The milk supply was normal and during test-feeding on the eleventh day of the puerperium 11¼ ounces of milk were secreted in 24 hours, averaging 2¼ ounces in each of five feeds.

Diffuse Round-celled Sarcoma of Uterus and Fallopian Tubes.

By A. C. BELL, F.R.C.S.

THE specimen was removed by operation from a woman, aged 38, who came to the out-patient department complaining of twelve months' continuous vaginal hæmorrhage, accompanied by a profuse and offensive discharge. Her past history was unimportant. Periods were regular up to the onset of the vaginal hæmorrhage; she had had one child, 20 years ago, and two miscarriages. On examination she was found to be anæmic and in bad general condition. There was a hard symmetrical mass which reached well above the umbilicus, and had been mistaken by her private doctor for a pregnant uterus. It was tender to touch, and appeared to be fixed. A provisional diagnosis was made of a degenerating fibroid, probably sarcomatous.

A pre-operative blood-transfusion was carried out, and the uterus, together with the tubes and ovaries, was removed by the abdominal route. The operation was very severe and was accompanied by much hæmorrhage from the enormously dilated blood-vessels. The patient died the same day.

An examination of the specimen showed that the cervix, the body of the uterus, and the fallopian tubes were infiltrated diffusely by a malignant growth which had sloughed into a considerably enlarged uterine cavity, the whole giving the appearance of a diffuse hypertrophy of the uterus. Pieces of tissue were removed for section from the cervix, fundus, and fallopian tubes, and microscopic examination showed that the tumour was of the nature of a round-celled sarcoma. The malignant cells are seen to infiltrate the muscle-wall of the uterus and, in one section, the wall of a blood-vessel. It is impossible from an examination of the specimen to say at what site, or from which tissue, the growth originated. There does not appear to have been any pre-existing fibroid in the uterus, but in view of the round-celled nature of the tumour, it is highly probable that the growth originated in the endometrium. Additional evidence for the endometrial origin of this tumour is obtained by examination of the fundus of the uterus, where one can trace an external coat having the appearance of uterine muscle, which is being invaded by malignant tissue from the cavity of the uterus.

It is chiefly on account of the remarkably diffuse nature of this growth, together with its size, that I have thought the specimen worth showing.

There is considerable literature concerning sarcomata of the uterus, and although much has been written in recent years, one of the most comprehensive studies of this condition was published by Piquand,[1] as long ago as 1905. This author gives a very complete review of 418 cases of sarcoma of the uterus. It was found that unlike carcinoma of the uterus, sarcoma of the body was five times as common as sarcoma of the cervix. There was a definite connexion between the existence of fibroids and the development of sarcoma in the uterus—a relationship which is well known. Apart from sarcomata arising in fibroids, Piquand divides sarcomata of the

[1] Piquand, 1905. "Le Sarcome de l'uterus; étiologie, anatomie, pathologie du sarcome du corps." *Rév. de Gynec. et de Chir. abdom.*, 1905, ix, 387-446.

body into those arising in the endometrium and those arising in the muscle, and in each case he subdivides them into localized and diffuse types of growth. This author has collected fifty-four endometrial sarcomata, of which thirty-three were of the diffuse variety, as seen in this specimen which his description would very well fit. He states that :—

" The uterus is enlarged by a diffuse endometrial sarcoma, giving the appearance of a symmetrical tumour, rounded or ovoid, much resembling a pregnant uterus. A section of the uterine wall shows that it is divided into two layers—an outer, having the characters of an ordinary uterine muscle, and an inner, consisting of a pale grey tissue which is soft and friable."

I should like to thank Mr. Bright Banister, under whose care the patient was, for his kindness in allowing me to show this specimen.

Discussion.—The PRESIDENT asked if metastases had been found after death. He agreed that certain sarcomata arose in fibromyoma, but he thought it was more usual for them to arise independently of these innocent neoplasms.

Mr. L. C. RIVETT said he was interested in this case as he did not believe that fibroids had any relation to sarcoma. It must be borne in mind that fibroids were present in about 10% of women and therefore were frequently found concurrently with other conditions.

Mr. BELL (in reply) said that there had been no post-mortem evidence of metastases.

An Unusual Cyst of the Uterus

By JOHN HOWKINS, L.R.C.P., M.R.C.S.

Mrs. H. W., aged 36, a multipara having had three normal labours,, was operated on for fibroids in 1932. There were classical symptoms and signs and at operation two fibroids were removed by myomectomy. The uterine cavity was opened and no intra-uterine fibroid polypi were found. Sterilization was performed by tying the tubes, and the uterus was ventrofixed.

In May 1934, the patient again complained of her original symptoms, which were menorrhagia and backache. The uterus was then removed by subtotal hysterectomy.

Specimen.—A globular uterus with a cervix 6 in. in length and about 4 in. diameter with a red polyp protruding through the os externum. The fallopian tubes had been removed at a previous operation. On sagittal section a cyst is seen to be present chiefly in the lateral wall, protruding into the posterior wall behind the endometrial cavity and containing 10 oz. of clear fluid. The cyst was surrounded by uterine muscle except in the lower part where it protrudes into the base of a polypoid mass springing from the left lateral and posterior walls of the canal. The polyp is 1½ in. long and 1 in. in diameter and is composed of tissue surrounding one large cyst and several smaller cysts. The endometrial cavity is enlarged and spread over the inner surface of the cyst; the endometrium appears to be slightly thickened. There is no communication between the uterine cavity and any part of the cyst. No myomectomy scar was seen.

Histology.—The microscopical section shows the walls of a major cyst and of a minor cyst; both are lined by epithelium which in parts is flattened cubical, and in other parts columnar, many of the columnar cells being ciliated. Beneath the basement membrane of the lining epithelium is a thin layer of connective tissue separating it from uterine muscle, which, in turn, separates it from the endometrium covering the base of the polyp. The uterine muscle appears to be normal and there is slight hyperplasia of the endometrium of the glands.

Discussion.—The possibilities of the origin of this cyst are :—

(1) That it arose from the interstitial portion of the tube which was tied at previous operation, the proximal end becoming occluded. The low position of the cyst is against this theory.

(2) That it is an endometrial inclusion-cyst comparable to an inclusion-dermoid.

(3) That it is a cystic dilatation of an undeveloped horn of a bicornuate uterus.

(4) That it is an endometrial implant in the myomectomy cavity. Against these last three are the facts that the cyst contained clear fluid and that no endometrial stroma was present beneath the epithelium lining the cyst.

(5) Cystic degeneration of an interstitial fibroid was ruled out by the presence of an epithelial lining to the cyst.

(6) That the cyst arises from some vestigial remnant such as the duct of Gartner, cysts of which are not uncommonly described in the lateral vaginal wall.

I have to thank the Bland-Sutton Institute for kind co-operation in this report.

The PRESIDENT said that Mr. Howkins had submitted a real problem. Clearly all the other forms of cysts in the uterus, apart from those arising from Müllerian implants or Wolffian relics, could be excluded, as Mr. Howkins had explained. The origin of a large cyst from a Wolffian relic within two years in a woman aged 36, would, however, surely be a unique event, especially in the absence of any virilizing hormone. This left an endometrial or tubal implant as the source. The fact that endometrial stroma was not present, and that, consequently, there was no blood, inclined him (the speaker) to the view of a tubal implant, such as Sampson had described in the ovary. This was a most interesting specimen.

Renal Cortical Necrosis Associated with Pregnancy

By R. K. BOWES, M.S.

RENAL cortical necrosis is a relatively rare condition of which there are now over 50 cases on record, the majority occurring in women and in association with pregnancy. Originally it was reported by Sir John Rose Bradford in 1898, and since then there has been a series of papers on the subject, including those of Rolleston in 1913 (describing 11 cases), Glynn and Briggs in 1915, Clifford White, 1918, Cruikshank, 1923, Crook, 1927 and, more recently, Davidson and Turner in 1930, and Kellar and Arnott in 1933.

Though almost exclusively a lesion confined to the female, Parkes Weber, in 1909, described it in a male aged 69, and Bamforth, in 1923, gave an account of a classical example in a man aged 37 with dysenteric symptoms.

In women it is commonly associated with pregnancy and its disorders, but there are on record cases occurring in scarlet fever, diphtheria, carcinoma of the stomach, and rupture of the liver.

The condition has for its main clinical signs almost complete suppression of urine, and this, coupled with a definite pathology, forms a dramatic complication of pregnancy.

The details of the case reported now are as follows:—

Mrs. H., aged 32, parity 2, was sent to St. Thomas's Hospital on October 27, 1933, by her doctor, four days after he had found albuminuria.

In the previous history there was no knowledge of renal disease or of a predisposing infectious fever.

The previous obstetric history was that the first pregnancy had been complicated during the seventh month by slight albuminuria and headaches which responded to medical treatment. The systolic blood-pressure was 140. Labour was six weeks premature in September 1932, and a macerated fœtus was produced. During the interval between the pregnancies the urine was free from albumin and the blood-pressure was normal.

The last menstrual period had been in March 1933. The patient, in view of the previous toxæmia, was extremely carefully watched throughout the present pregnancy, her doctor testing the urine at weekly intervals. She was well and albumin-free till within four days of admission to hospital. It was then that she noticed swelling

of the ankles and lower legs, and the urine was found to contain albumin. Headaches and indistinct vision occurred and she was sent to St. Thomas's Hospital.

On admission she was noticed to be very apprehensive and fidgety. There was no jaundice. The ankles and lower legs showed a moderate degree of œdema; there was no œdema of the face. The blood-pressure was 140/80, and the urine contained a large amount of albumin. The uterus was about two inches above the umbilicus and the child was alive.

She was put on to protein-free diet and went into premature labour the same evening, delivering herself spontaneously of a still-born fœtus, weighing 4 lb., and displaying no obvious abnormalities. Her condition gave rise to no anxiety. The following day, October 28, about twelve hours after the labour, she passed 12 oz. of urine, which contained 5 % of albumin. On October 29, as she had passed no further urine, a catheter was employed and two drams of clear urine were obtained. She was not at all drowsy, but was easily excitable; the blood-pressure was at its previous figure. Treatment by administration of diuretics and glucose saline was begun. On October 30 four drams of urine were passed and the patient was becoming by turns drowsy and delirious. From October 30 to 31 the drowsiness increased, though the skin was moist and the tongue comparatively clean. Vomiting developed and there was general muscular hypersensitivity. Epigastric pain and tenderness were also noticed. The patient died suddenly on the night of October 31, but had had no fits.

The biochemical features may be summarized as follows: The so-called urine contained no urea (by the hypobromite test). It was sterile on culture. In one specimen there was a small plug of fibrinous material which made a diagnosis definite. The blood-urea and creatinine were both raised, the former being 160 mgm. % and the latter 6·4 mgm. %.

Report on post-mortem examination.—There were no external features of interest. There was œdema of the subcutaneous tissues of the legs.

The kidneys were slightly enlarged. The capsules stripped easily, revealing underneath, a yellow cortex stippled with hæmorrhagic areas. There was typical necrosis of both cortices with narrow areas of hyperæmic tissue under the capsule and near to the medulla, the latter being of a deep red colour, though areas of necrosis are seen here as well. The suprarenals were normal as also were the stomach, intestines and spleen.

The liver showed diffuse cloudy swelling, but no necrotic or hæmorrhagic areas.

The uterus was bulky and had a softened cervical fibroid the size of a cocoanut in it.

There was œdema of both lungs and of the aryepiglottic fold. The pericardium was normal, but the myocardium showed cloudy swelling.

Microscopical examination of the kidney.—There is some œdema of the capsule; underneath it are hæmorrhagic and congested areas. The convoluted tubules show marked changes, the cells being swollen, the nuclei disintegrated and the lumina filled with granular material. Many are totally necrotic. The glomeruli are congested and stain poorly, many nuclei failing to stain at all. There is no proliferation of the endothelium.

Interstitially there are areas of hæmorrhage and polymorphic infiltration. Several of the vessels are choked with thrombi near the glomeruli. All these changes are rather patchy in distribution, the areas less affected showing cloudy swelling. The medullary tubules are also involved, many being filled with hyaline material.

The following points are of interest with regard to this case: (1) The previous history of a pregnancy toxæmia; (2) the fact that excellent ante-natal supervision had obviously no effect on the incidence or advancement of the condition; (3) the rapidity of progress; (4) the patchy nature of the pathological changes which appears to indicate a thrombotic origin; (5) the appearance in the urine of a plug of fibrin which is probably allied to the fibrinous material seen in the tubules.

My thanks are due to Dr. J. P. Hedley and Professor Dudgeon for permission to report the case and show the specimen, and to Dr. N. Jackson for the notes on the previous pregnancy.

Discussion.—The PRESIDENT said that he had known a case in which cortical necrosis of the kidneys in pregnancy had led to the death of a patient who had been successfully treated with lead for intestinal sarcoma a few years previously. Previous to pregnancy, no sign of lead-nephritis had been noted, but no doubt the kidney had been damaged. The condition was not associated with eclamptic phenomena.

Mr. CLIFFORD WHITE said that in 1919 he had read a short paper before the Section on two cases of puerperal anuria in which the renal capsule was incised and portions of the kidney substance removed for examination.[1] He regarded these cases as being in the early stages of what was called cortical necrosis in the post-mortem room. The clinical history of both was very similar to the clinical history in the present case. Briefly, the histories were: The first case was in a primigravida, twenty-four weeks pregnant, with intra-partum eclampsia, and grossly œdematous all over. She was delivered by Cæsarian section, the convulsions immediately ceasing and consciousness being regained in two hours. For the following nine days she had almost complete anuria, only passing about 2 oz. of urine a day. There were no symptoms of uræmia. On the ninth day he (the speaker) had exposed the kidney through the loin and incised the capsule. During the next eight hours the patient passed 6 oz. of urine per urethram, besides what escaped through the drain in the loin. The next day 38 oz. were passed containing a quantity of leucocytic casts. The patient rapidly made a complete recovery.

In the second case recorded, the patient was in the sixth month of her second pregnancy. She was delivered by induction of labour because of severe albuminuria and generalized œdema. After delivery, the œdema increased and she never passed more than 9½ oz. of urine in each twenty-four hours. The kidney capsule was incised; she passed 20 oz. the next day and rapidly recovered. Small portions of the kidney substance were removed at the operation, and sections showed marked dilatation of the renal tubules, which was shown in the illustration of his (the speaker's) original article in 1919.

In 1927, Mr. Arthur Crook had read a communication on a case of necrosis of the kidney in which the kidney was incised on the fifth day and the patient recovered.[2] In the discussion on that paper, he (the speaker) had put on record a case, under the care of Mr. Miles Phillips, which appeared to be clinically a typical case of cortical necrosis of the kidney. Only four drams of urine were passed in twenty-four hours. On the fourth day the capsule of the kidney was split longitudinally. Forty-eight hours after the operation the patient was passing four pints of urine daily, and she made a perfect recovery.

Cases of puerperal anuria were so rare that it was doubtful if anyone's personal experience would extend to more than two or three, and he (Mr. White) therefore wished to draw attention to the fact that in these four cases the patients had recovered after incision of the kidney capsule. All four were clinically similar in every way to recorded cases in which, after death, the patients were proved to have been suffering from cortical necrosis. He therefore suggested that in cases of puerperal anuria it would be better to incise the capsule of the kidney within the first day or so of the anuria, and allow the kidney substance to bulge through the incision and relieve tension inside the capsule, than to wait until cortical necrosis supervened.

Concealed Accidental Hæmorrhage treated by Cæsarean Hysterectomy

By W. C. W. NIXON, F.R.C.S.

THE specimen is the result of a Cæsarean hysterectomy performed on account of concealed accidental hæmorrhage. Briefly, the clinical history is that the patient was a primigravida aged 42. At the sixteenth week of her pregnancy, Mr. L. C. Rivett removed two large fibroids. At the thirty-second week the patient complained of severe abdominal pain and slight loss of blood. On admission to hospital she was found to be shocked and suffering from concealed hæmorrhage. There were no signs of toxæmia except that the urine had a heavy cloud of albumin. The blood-

[1] *Proceedings*, 1919, xii (Sect. Obst., 27).
[2] *Proceedings*, 1927, xx, 1249 (Sect. Obst., 27).

pressure was 140/90. Conservative measures were adopted and the shock was treated. Owing to the failure of this line of therapy to arrest the internal bleeding, operation was decided upon. On opening the uterus, the placenta was found already separated by a large retroplacental clot. A macerated fœtus was removed. Owing to the inability to control the uterine bleeding, hysterectomy was performed. The patient made an uninterrupted recovery.

Pathological description.—On examination of the gross specimen there will be seen a fibroid at the level of the internal os, and other seedling fibroids. On the cut surface there are large areas of hæmorrhage which contribute to the unusual thickness of the uterine wall. Beneath the peritoneal surface also there is hæmorrhage.

Microscopical appearances.—The usual stains were employed, including one for fat. No fatty degeneration or focal necrosis of the muscle is present. As a control, a small piece of uterine wall, removed at the time of a Cæsarean section for disproportion, has been used. There is much œdema separating the muscle-bundles and this becomes less the nearer one approaches the decidua. One interesting feature is the marked vacuolation of the muscle-fibres just beneath the peritoneum. That this is not due to fat has been proved. As an alternative one might suggest that this was due to glycogen. This is only a tentative suggestion as no examination for glycogen has been made. Intra- and inter-fascicular hæmorrhage is obvious, but a point of interest is that it is practically localized to the outer half of the uterine wall and is almost entirely absent from that part of the wall beneath the decidua. There is swelling of the endothelial cells of the blood-vessels but leucocytic infiltration is not excessive. Microscopic examination also reveals another difference from the normal, namely, the marked decidual reaction beneath the peritoneum. There appear to be a series of decidual buds which are absent in the section from the normal uterus.

When the muscle-fibres are counted per mm. of field it is found that the number in the outer zone of the uterine wall is on an average 18·01, whereas in the inner zone the number is 30·05, a figure which approximates to that which one finds in a normal uterus at term. This disparity is to be expected, owing to the vast amount of hæmorrhage found in the outer half of the uterus in this specimen.

Summarizing the findings in this case, one is impressed with what appears to be a definite demarcation of the uterine wall into an outer half which is grossly pathological, and an inner half which seems to be normal, the former revealing massive inter- and intra-fascicular hæmorrhage of the muscle, vacuolation of the muscle and a decidual reaction, in contradistinction to the latter, which is conspicuous by the absence of any gross pathological changes.

I must express my thanks to Mr. Rivett for allowing me to show this specimen.

Discussion.—The PRESIDENT said that Mr. Nixon had stated that there was "no toxæmia" in his case, but as there was albuminuria and the blood-pressure was raised, he (the President) thought that there must have been some toxæmia, and that this was due to the degenerated fibromyomata which had been removed.

Mr. L. C. RIVETT said that the fibroids removed were markedly degenerated to a fluid degree and he did not think they were infected.

Section of Laryngology and Section of Otology

COMBINED SUMMER MEETING

HELD AT BIRMINGHAM, JUNE 8 AND 9, 1934

[*June* 8, 1934]

Chairman—MUSGRAVE WOODMAN, M.S. (President of the Section of Laryngology)

Organisms in Chronic Bone Infections of the Sphenoid

By F. A. PICKWORTH

(*Director, Joint Board of Research for Mental Disease, Birmingham*)

BACTERIOLOGICAL culture methods of saline wash-outs from the sphenoidal sinuses of Birmingham Mental Hospitals patients indicate that the chances of finding organisms in any given case are 3 : 1, and of the organisms found, 40% will be streptococci, 50% staphylococci, 30% diphtheroids; other organisms in proportion. Similarly, of swabs taken from the sphenoidal sinuses within an hour or two of death, only one in four will be found sterile, and further investigation will show 50% streptococci, 50% staphylococci, 30% diphtheroids and 17% *B. influenzæ*. Of eight cases, where small portions of bone removed from the posterior wall of the sphenoid with aseptic precautions were examined by bacteriological culture, only two were sterile; five showed streptococci, four staphylococci, one diphtheroid and two *B. influenzæ*. There can be no doubt that these organisms are actually present in the sinuses, but their clinical significance presents some difficulty. The only reliable method of determining whether such actually grow in the tissues or whether they are chance atmospheric deposits, is the examination of histological sections and the detection of the organisms in situ.

In carrying out this research, an important fact was first overlooked, namely that the organisms which exist for years under unsuitable conditions for rapid growth, cannot be expected to show the geometrical regularity exhibited by young organisms growing in an unlimited volume of rich culture media as is usually seen in the laboratory. Very little work has been done on organisms which are grown under unfavourable conditions, but it is known that the diphtheria bacillus so grown develops an incredible number of different shapes and sizes. Under the influence of acid or alkaline reaction of the media the influenza bacillus also shows bizarre forms.

In common with the method of other workers, the search for organisms was originally restricted to the finding of geometrically perfect shapes. The object of this paper is to point out that such organisms do not represent the chronic form of organism which is growing in the tissues but are all *recent extensions* from these lesions. Such organisms are typified by fig. 1, showing organisms in a pituitary gland

vessel—histologically proved to be a recent extension from a chronic sinusitis [1] ; fig. 2 illustrates organisms in the sheath of the optic nerve which are recent extensions from streptobacilli and Gram-negative cocci existing in a chronic

FIG. 1.—Diplococci in pituitary gland.

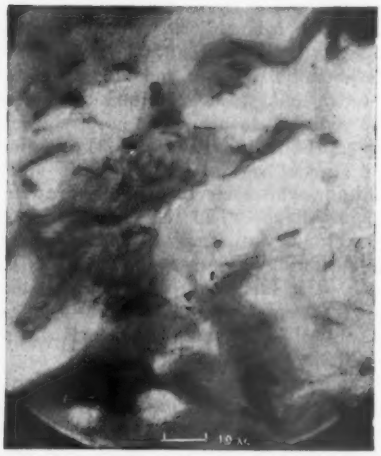

FIG. 2.—Diplococci in optic nerve. From the *Journal of Laryngology and Otology* [2]

sphenoidal infection [2] ; and fig. 3 shows organisms in the perivascular tissues of the carotid artery in the sphenoid. The latter are, in relation to adequate oxygenation and nutriment, analogous to a large volume of rich culture media, and

in consequence, show the shape and regularity of a row of billiard balls. The recognition of such organisms presents no difficulty, but this very fact also indicates that they are essentially *young* forms of organisms, probably of only a few hours growth.

A sphenoidal sinus (fig. 4) was selected for histological investigation of organisms because the chronic bone changes surrounding the sphenoidal sinus indicated a very long duration of the infection, and pus, containing cocci, was present both in the sinus and above the pituitary gland. The man died of pneumococcal meningitis, and it is quite apparent that the chronic infected sinus was directly related to the

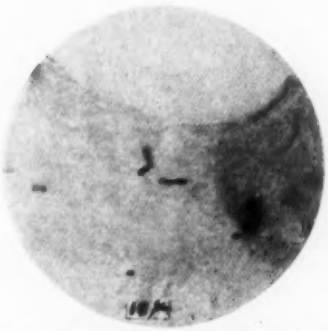

FIG. 3.—Streptococci in carotid artery. From the *Journal of Mental Science* [12].

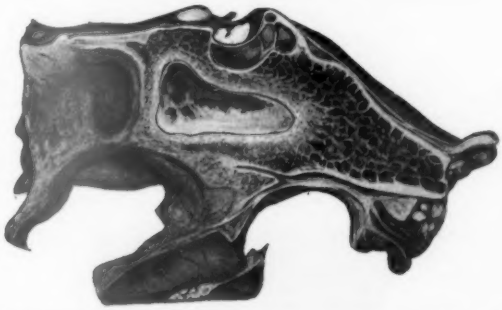

FIG. 4.—Chronic sphenoidal sinusitis. Bone changes, pus in cavity and over pituitary gland. From *Zeitschrift f. d. Gesamte Neurologie und Psychiatrie* [13].

cause of death. Irregular Gram-positive filaments were occasionally seen, and it is possible that had more sections been examined, we might have come across a few regular cocci in the bone, but as the main object was to find the causal organisms of the chronic, as distinct from the acute, condition, it was disappointing that a case which macroscopically seemed so definite showed no recognizable organisms in the bone.

The solution of the problem was afforded by another case [14] in which a chain of streptococci (fig. 5) just below the sphenoidal sinus epithelium presented distinctive differences from the regular appearance seen in presumably younger growths of the same streptococcus growing on the surface of the epithelium.

Instead of the expected geometrical regularity there are (*a*) triangular, (*b*) club shaped, (*c*) bacterial forms of the organism, and (*d*) irregular spacing in the filament. At a greater distance below the surface the same streptococcus (fig. 6) showed an irregularity of the Gram-positive elements in the filament, which was so marked that, unless the intermediate forms had been observed, one could not identify them as streptococci.

We were thus able to follow all stages between young perfect streptococci and the chronic inhibited form of filaments found in the bone of the sphenoidal infection

FIG. 5.—Streptococci under mucosa. From the *British Medical Journal* [14].

FIG. 6.—Streptococci near bone. From the *British Medical Journal* [14].

illustrated by fig. 4. With this experience we searched specially for filaments containing Gram-positive granules in other cases. This was done in 200 cases by decalcifying thinly sawn pieces of sphenoidal bone in 5% trichloracetic acid saline, then staining paraffin sections by Gram's stain; in 80 cases, i.e. 40%, filaments containing Gram-positive granules were detected. Fig. 7 shows such typical filaments in the sphenoidal bone.

The mutation of organisms has lately occupied much attention. Dochez and others (1932) [3] have recorded observations which suggest that the occurrence of

"colds" may be associated with change of avirulent to virulent types of influenza bacilli. Many pathological organisms hitherto considered specific alter during culture or passage, so that the resulting organism is of an entirely different type from the original. *Bacillus typhosus*, for instance, will produce four types each with different characteristics. Todd (1927) [4] and others have shown that virulent cocci can be inhibited by growing them in immune serum, the resulting cocci having different cultural characteristics and pathogenicity from the original. The same author (1930) [4] showed that, by cultivation under conditions of peroxidase formation, cocci could be reduced in virulence by about a thousand times. The avirulent strain is more resistant to anti-bacterial ferments (such as are present in human tears) than the virulent. Reactivation of the virulence could be accomplished by growing under conditions of maximum oxygen tension without peroxidase formation. Arkwright (1921) [5] and others have shown that organisms when grown in immune sera change from virulent "S" types to the non-virulent "R" types; he says: "In spite of the lack of virulence of the 'R' form, it does not appear to lack vitality and vigour for vegetative growth." Dawson and Avory (1927) [6] found that "rough" pneumococci could be reverted to the "smooth" form by subculture in the serum of animals immunized with "rough" pneumococci. Since normal serum usually contains sufficient antibodies to "rough" pneumococci

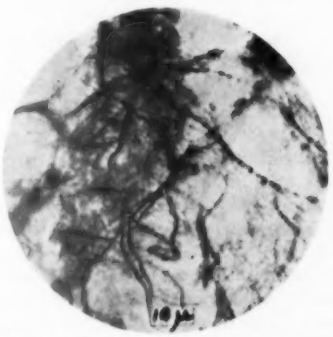

FIG 7.—Streptococcal filaments in bone. From the *Journal of Mental Science* [12].

to cause reversion as indicated above (Dawson 1928) [7], similar changes might be expected with virulent streptococci; also we have in this change an explanation of the widespread damage of distant organs which may occur, due to a focus of non-virulent (but potentially virulent) organisms.

Parish and Okell (1928) [8] found, as a result of injection of animals with virulent streptococci plus sufficient anti-serum to prevent a fatal septicæmia, that there was a "subacute infection towards the end of the first week, or later, characterized mainly by arthritic lesions, from which the hæmolytic streptococci can readily be recovered." Watkin and McKenzie (1929) [9], by culturing fragments of bone removed at operation for osteomyelitis, found organisms at a distance of at least one inch from the seat of manifest disease. Logan Turner and Reynolds (1931) [10] published a case of double infection in which chronic streptococcal sphenoidal sinusitis was followed by meningococcal meningitis and death; there were also acute streptococcal changes in the sinus membrane, bone, cavernous sinus, fifth nerve and pia mater. These acute changes could only reasonably be explained by the reactivation of an avirulent strain of the streptococcus, following tissue damage and diminished resistance of the host caused by the meningococci. An

important case has been published by Rosenow (1927) [11], which clearly demonstrates the type of pathology related to our subject; the patient had chronic cystitis, sphenoidal sinusitis, and atheroma of brain vessels; trauma of the bladder insufficient to cause harm to a normal individual, set up a septicæmia, which caused fatal thrombosis of the cerebral arteries.

It is submitted that the filaments demonstrated represent inhibited streptococci which are actually growing in the sphenoid bone, and that the patient has a high antibody content in the blood to the particular organism with its virulent prototype. The reversion of the chronic form to the virulent presents many interesting problems. That organisms do actually exist in a viable state in bone infections is clinically recognized by surgeons who hesitate to open up any long healed chronic infection because of the danger of setting up an acute local infection or even a septicæmia by distributing the clinically dormant organisms.

The long duration of chronic infections of the nasal sinuses presents a marked contrast to systemic infectious diseases. Cases in which sinusitis existed over a period of ten years have been recognized. The macroscopic bone changes often found are evidence of a persistence of infection over many years; the associated clinical symptoms of one case suggested an infection lasting twenty-five years.

An illustration is given (fig. 8) showing pus in the spheno-ethmoidal recess of a child only 3 months old. The pus was the most striking macroscopical lesion

FIG. 8.—Pus in spheno-ethmoidal recess of a baby three months old.

seen post mortem; the clinical condition and cause of death were, however, ascribed to gastro-enteritis. Even at this early age sinus infection is an important cause of systemic disease and death; in recoverable infections the child forms antibodies to the organism, thus fulfilling the conditions by which a virulent streptococcus mutates to an avirulent type suitable for permanent establishment in the bony tissues of the skull. Such early bone sepsis needs correlation with irregularities in the development of the sinus as well as with the physical and social development of the individual.

A recognition of the fact that irregular Gram-positive granules within filaments seen in bone sections may be forms of inhibited streptococci, would materially assist research on bone sepsis and on the incidence and complications of nasal sinusitis.

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P. WATSON WILLIAMS said that members could not fail to realize the importance of this investigation of the sinuses; the slides which Dr. Pickworth had exhibited represented years of patient investigation. Of great importance was the peculiar chronicity of these conditions, the organisms penetrating the bone and other tissues and remaining more or less active, or at least ready for reactivation after long periods of quiescence. It was the clinical aspects, however, with which laryngologists were particularly concerned. There were many more cases in which the clue to successful diagnosis and treatment lay in the sphenoidal sinus region than was generally believed. Dr. Pickworth had shown that after the acute stage, the infection might persist for years, and could do infinite mischief. Cases of sinus infection in the child often presented systemic reinfections elsewhere. Probably when a definite infection became chronic the patient never threw it off completely, and it was liable to become reactivated even after a number of years. He (the speaker) wished that there had been time to refer to cases which showed the effects of reinfection; for example, he had intended to speak on cases of Frölich's syndrome in young boys, in which sphenoidal sinus infections were present and sinus disinfection produced favourable results.

Sphenoidal Sinus Disease in Mental Disorder

By T. C. GRAVES

My contribution is an account of two cases,[1] representing a large number, to illustrate the continuation of that work on nasopharyngeal sepsis in mental disorder which has already been reported.

In these cases modes of treatment other than those having an action on the septic state are without avail on the psychosis; thus, the sedative drugs fail to remove the excitement of mania arising from closed foci of sepsis.

In the two cases now reported definite disease was present in the sphenoidal sinus and in both there was serious circulatory disturbance. The first was one of acute mania with cardiovascular intoxication, the second was one of mental confusion with optic neuritis and atrophy and other symptoms suggesting pituitary disorder—at one time suspected as pituitary tumour—and myocarditis. In both cases medicinal measures were without effect on the psychosis and associated sensory disturbances but these were ameliorated and recovery ensued following surgical treatment of the nasopharyngeal sepsis, aided by non-specific therapy to provoke a focal reaction and so stimulate discharge of residual sepsis after drainage.

CASE I.—SEVERE ACUTE MANIA

Female, hairdresser, aged 25. First certification on admission to mental hospital, April 12, 1933. Discharged November 3, 1933. Recovery maintained.

History.—Since age of 21: Influenza twice, nasal catarrh and later cacosmia and cacogeusia, also headaches, principally right temporo-parietal, but also bi-frontal—at first one day a week, later continuous—and two nervous breakdowns, one of five months' duration. In 1932 patient was at work, but in autumn and ensuing winter depression, asthenia, and malaise appeared and intensified. She ceased work and lost weight. In March 1933, severe abdominal pain with collapse was followed by undue excitement and unnatural elation by day and night. On admission to mental hospital there were noted, severe acute mania, variable confusion, faulty habits, anæmia, cardiovascular instability, general asthenia, and dental sepsis. The nasal passages and tonsils contained pus.

Course and treatment.—Terebene intramuscularly and a variety of sedative drugs by mouth and injection were given during April without improvement, in fact, digitalis, strychnine, brandy and oxygen were frequently required to combat circulatory enfeeblement and collapse. Early in May anti-scarlet serum 50 c.c. was

[1] Shown at the meeting held on June 9.

given, and after this the patient was stronger and co-operated better. Sedatives were given but no collapses occurred. She even tolerated dental treatment of four extractions under general anæsthesia. General anæsthesia for a sinus examination on May 30 was not well tolerated but it was possible, by Watson-Williams technique, to examine the six sinuses. Pus was found in the right sphenoid and left antrum, and these were drained intranasally.

A course of non-specific therapy, using T.A.B. vaccine, was given. The patient was still maniacal and sedative drugs were still necessary as temporary palliatives, but the circulation was stronger.

On August 1, under general anæsthesia, septic tonsils were removed by dissection, and from August 8 on alternate days a course of four intramuscular injections of colloidal sulphur was given. From then onwards the possibilities of recovery became more manifest, for after July sedative drugs were not required. The weight which had fallen from 7 st. 6 lb. to 5 st. 13 lb. began to rise and at the end of August was 6 st. 2 lb. A further course of colloidal sulphur was given during September, and following this convalescence was assured. At the end of October the weight was 8 st. Patient now recognized that she had been mentally ill and was able to describe her experiences, including the development and, since the operations, the cessation of the sensory disturbances and catarrh. Objectively she was more stable, less irritable and more sociable. General muscle tone improved, shown in gait, carriage, and facial aspect and mobility. Facial colour replaced pallor. She was discharged from the mental hospital on November 3, 1933. She has remained well since then and has returned to work.

Case II.—Mental Confusion, Optic Atrophy, Myocarditis, Dyspituitarism

Housewife, aged 41. First certification on admission to mental hospital on August 9, 1929. Discharged April 12, 1934.

History.—Frequent severe headaches and influenza. Five normal pregnancies. Increasing weakness during last two years. During sixth pregnancy developed obesity, enormous appetite, enlarged thyroid and tremulousness, cyanosis and vertigo: periods of confusion and anger alternated with sleepiness. After delivery in March 1929, this condition became worse, visual acuity was reduced and headaches were severe. Myocarditis was diagnosed and pituitary disorder suspected; sugar curve of pituitary type. No tumour found on radiography. Treated with pituitrin injections. Mental state became worse and patient was admitted to mental hospital.

Condition on admission.—Very feeble health, low blood-pressure and unstable pulse. Grossly obese. Purulent conjunctivitis and blepharitis. Pupils reacted normally, impossible to examine fundi. Wassermann reaction negative in blood. Confused, depressed, resistive, noisy and restless. Visual and auditory hallucinations. Perforation of both tympanic membranes and evidence of sinus and tonsillar disease.

Course and treatment.—Circulatory instability was increased by a profuse menstruation but by the end of the month she had rallied and her mental state appeared to be that of secondary dementia with hallucinations and excitement, for two years and a half. Amenorrhœa now followed and her condition during that time was such that only medical treatment could be given. She had sedatives, pituitary, ovarian, and parathyroid injections and tablets, tonics, potassium iodide, and gynæcological treatment. She lost 5 st. 2½ lb. and there was little change in her mental state; instability of equilibrium became more marked and she constantly fell to the right. With great difficulty her eyes were examined and bilateral optic atrophy suggestive of pituitary pressure was found. Her pulse at this time was 60. Lumbar puncture gave normal cerebrospinal fluid, not under increased pressure, although immediately after puncture she walked without falling. The findings now suggested that the pituitary damage was due not to tumour but to local toxæmia.

June 22, 1932. Nasal sinus examination revealed infection of both sphenoids (*B. influenzæ*), especially left. These sinuses and the antrum were drained intranasally. The patient improved mentally, becoming more intelligent and quieter, and sedative drugs were no longer necessary.

November 29, 1932. Both septic tonsils were removed by dissection. Patient was now rapidly gaining weight and a definite mental improvement was apparent. She was quieter and more composed, and she slept well and was able to help in the wards, but during her menstrual periods she became confused, noisy, and irritable. Although she was steadily improving, blepharitis, and sclero-conjunctivitis were now more marked than during the period of her acute illness and persisted in spite of adequate local treatment, especially on the left side. On November 28, 1933, another sinus examination was carried out. The antrum was clear but the sphenoidal and ethmoidal sinuses contained flakes of mucopus.

A similar procedure on December 19 and other dates was followed by improvement in the conjunctivæ and lids.

By this time the menstrual relapses were much less marked and were characterized by simple irritability with no mental confusion. She went home on leave, worked intelligently in the hospital laundry and was able to discuss her case with insight; this improvement continued. She was discharged April 12, 1934, and has remained well at home.

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P. WATSON WILLIAMS said that Dr. Graves could not be accused of rushing into the sinus treatment, as one of the patients had been a sufferer from the mental condition for over three years, and it was only his (Dr. Graves') feeling that the origin of the trouble might be found in the sinuses that had made him determine to investigate and treat the sphenoidal and other sinus infections present.

What struck those who saw cases of sinus disease not in asylums was the necessity, if possible, of seeing and treating these patients at an earlier stage. Failing this early recognition and treatment, there were many who would drift into lunacy, or something nearly as bad.

On the Control of Air-Pressure in the Lungs after Tracheotomy

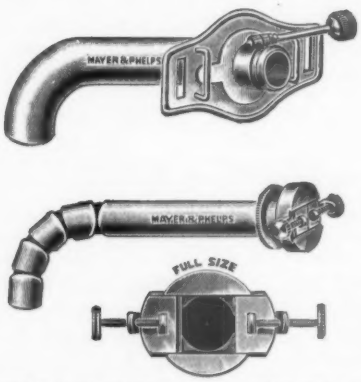
By MUSGRAVE WOODMAN (Chairman)

WHEN I was a house-surgeon I was impressed by a case of chronic intestinal obstruction with superimposed acute obstruction, the abdomen being much distended and the coils of intestine showing through the skin. The patient was much distressed, and he had a poor pulse. My chief carried out a colostomy and tied in a Paul's tube of efficient diameter, and out of this came large quantities of fæcal matter. At first this brought great relief, but with the passage of the fæces, anxiety increased, the pulse diminished, and within twenty-four hours the patient was dead. That impression has remained with me.

Three years ago a man came to me with chronic obstruction of the larynx; he had had this with increasing severity, for three or four months. He entered hospital, and we inserted a good tracheotomy tube, of wide bore. The relief from this was instantaneous, but lasted only for a few hours, at the end of which he began to get water-logged. He had a cough, and mucus and saliva streamed out of the tracheotomy opening. Œdema and collapse of the lungs developed and the patient died. His death was due, not to pneumonia, but to complete collapse of the lung.

I have studied that case and similar ones, with the help of Professor Daly. It is evident that we must control the pressure in the trachea, just as we should, and probably do, control the pressure in colostomy when there exists considerable pressure inside. Mr. Negus has shown us that the primary function of the larynx is sphincteric; it is not vocal. The question arises whether we are too abrupt in cutting out, by the tracheotomy opening, that sphincteric control which is the necessary and normal part of human physiology.

For that reason I have been using in my practice for two years, a valve of a simple character, designed to control this intratracheal pressure. It fits on to a Durham tube, and the size of the outlet can be readily adjusted by the nurse, but the outlet is never as large as the tracheotomy tube opening. The patient still has to work for breath, but less than before, and I instruct the nurse to give the screw a turn every hour, so as to gradually widen it to the extent that it ought to be widened. It can be removed from the inner tube for cleaning. The great importance of the valve is that it must be used immediately after the operation, when the distress due to the change is at its greatest.



At the top of the illustration is an ordinary Durham outer tube. In the centre is the inner tube with the special valve locked in position. The lowest figure shows the detached valve with the flanges in the fully abducted position.

Discussion.—V. E. NEGUS said that the larynx played an important part in controlling the inlet and exit of air into and from the lungs. He himself had designed a valve in this connexion which, however, was not satisfactory, and he was interested to see one that had proved to be successful. The question of washing out of carbon dioxide must be considered. Occasionally a patient underwent an apparently successful tracheotomy and subsequently died. The reason, he thought, was that the alveolar carbon dioxide in laryngeal obstruction rose from the normal 5·6%, even to 8%, and the alkali reserve had to be raised in proportion to fix the higher percentage of carbon dioxide in the blood. If tracheotomy was performed, the patient might, even in a few breaths, bring the alveolar carbon dioxide down to 5·6%. The lowered percentage of carbon dioxide in the blood was fixed and was not sufficient to stimulate the respiratory centre, which was set to a high level. The drop of this percentage and its fixation by the excess alkali reserve led to cessation of respiration, and possibly to death. The immediate indication was to give carbon dioxide. The valve would be of use in this respect if applied at the danger time, immediately after the tracheotomy. If springs could be fitted to Mr. Woodman's valve to control the size of the artificial glottis it would be an advantage, because the glottis opened at inspiration, and partially closed at expiration.

W. STIRK ADAMS said that this subject involved two problems. The first concerned the period immediately after tracheotomy. He could visualize two cases: In one there was acute œdema of the lungs. A child, aged 5, with obstruction from laryngeal diphtheria underwent

an emergency tracheotomy, without anæsthesia. Blood-stained mucus poured out of the tube for twenty minutes after the operation, and the condition then subsided, with recovery of the patient. That was with a free tracheotomy tube. In the other case, laryngeal obstruction followed an influenzal acute upper respiratory infection. A man, aged 30, developed dyspnœa during the night, and when seen at noon on the following day he was conscious but very distressed. He was propped up in bed, ashen grey in colour, and using all his accessory respiratory muscles; he tried to take his false teeth out and fell back unconscious. He (the speaker) had performed tracheotomy immediately, but although respiration was then free, the patient rapidly became water-logged and died twenty minutes after the operation.

The other problem concerned what might happen three or four days after operation, in cases of more chronic obstruction, in which there was a secretion of infective mucus.

The CHAIRMAN (in reply) said that he would be glad to try improvement on the lines which Mr. Negus had suggested. He again emphasized the importance of using the valve immediately after the tracheotomy; it was kept in readiness in the theatre for connexion with the inner tube.

Speech Re-education Methods

By FREDA PARSONS

I HAVE been asked to give a short demonstration of my methods in the teaching of phonetic speech sounds, especially in relation to patients with cleft palate and delayed speech.

The first essential in all this work is to arouse in the patient a keen interest and a joy in correct speech-formation. Before treatment a large majority of patients are mentally depressed owing to the realization of their speech inability, and this depression develops into a very sure sense of inferiority.

Speech training is necessary for all who find they are unintelligible through speech, according to the language in which they are speaking. Hindrance to normal speech is serious, for three main reasons: (1) Speech is the ventilator of the mind, and when normal speech formation is impossible or difficult, the personality becomes imprisoned. (2) Speech has been well described as "the trellis on which the mind can climb"; so that when speech is difficult the mind has no trellis, and mental development is hindered. (3) Speech is the chief means of social intercourse. If children, or even adults, are unable to speak they feel socially and otherwise inferior.

Speech-training must therefore give intelligibility to speech. I have not time to show my methods for people who stammer, or for those who have aphasia and similar speech defects. I shall merely show some exercises which I use in the treatment of cleft palate and delayed ability to speak.

I not only teach the formation of sounds but also the oiling of the speech machinery, thus giving it the flexibility necessary for the formation of ordinary voiceless stops and voiceless continuants. Then we pass to the teaching of tone-placing, and the importance of exercising the right "pull." The vowels will then fall into shape normally. For that reason we pay little attention to vowel practising; we concentrate on consonants, and indulge in conversational games.

I try to instil into my pupils a sense of confidence and ability and get them to take joy in this game of speech. I arrange different exercises every week to keep up the interest and excitement. Especially I get them to produce the sound through the mouth, not through the nose.

[Among the pupils demonstrated was a lady, aged 56, who had a complete cleft palate; she was wearing an obturator, and she had been unable to ring up anybody on the telephone, or to make herself understood in business. Her enunciation at the demonstration was not far short of perfect, and she had been able to resume her business without inconvenience.]

Discussion.—F. B. GILHESPY: In a discussion by the Section of Otology in 1932 on the Prevention of Deaf-mutism, I pointed out [1] that the pitch of the consonants was mainly situated in the region of 8,192 double vibrations a second (C_8 tuning fork), and that the vowel sounds were situated lower in the scale between 400 and 2,400 d.v. a second. A table from Harvey Fletcher's book, "Speech and Hearing," gives the frequencies of the various vowels. If the hearing below 2,000 d.v. is lost, then there is a great confusion in distinguishing certain vowels, apart from difficulty in hearing them, as certain pairs of the vowels have fundamental notes which are so close together that differences in frequencies would not be appreciated unless one of the pair possessed an overtone by which to recognize it; these overtones lie between 1,500 to 2,000 d.v. per second. It has occurred to me that the table given is too rigid as there must be some difference in pitch between the voices of males and females or soprano and baritone. This is the case, but is not very great in the spoken voice. In discussing this with Miss Parsons, she referred me to Aikin's scale, which I here reproduce, and this I think shows more clearly than any table the overtones used to distinguish vowels whose basal notes show very little difference in frequency. By knowing the pitch of the vowel AH in oneself, it would appear possible to know with what pitch we are testing patients when using vowels and consonants for the purpose. People with nerve-deafness fail first on their consonants. I must warn the members that after I had asked a patient suffering from nerve-deafness for a fee of three guineas, he, in good faith, handed me two; the mistake is explained by reference to the table and scale.

AIKIN'S RESONATOR SCALE

OO	OH	AW	ŏ	AH	ŭ	ER	ă	ĕ	EH	ĭ	EE
Who	knows	aught	of	art	must	earn	and	yet	take	his	ease
400	500	600	700	825	1000	1500	1800	1900	2100	2200	2400
					475	500	750	550	550	450	375

(Figures represent characteristic frequency of the vowel sounds.—FLETCHER.)

A simple experiment demonstrates phonetically that we use the overtone to distinguish OO and EE. If these vowels are whispered with the ears blocked, they cannot be distinguished by the speaker, as only the low tones produced in the pharynx are heard, while with the tongue raised in the mouth, the high overtone of EE is projected through the lips. The facts which I have mentioned are not new ones, but they have practical importance in teaching the deaf, and possibly may find some application for us in routine testing of deaf patients. It appears germane to this discussion to point out how little is taught of phonetics in our standard textbooks on ear and throat work, apart from the remark that a patient must rest his voice or an accusation of faulty voice production.

E. A. PETERS referred to an instrument, the multitone, one of which would be exhibited during the next session. It had an ingenious arrangement, whereby, in case of deafness, it would be possible to reinforce either overtones, or the fundamental lower tones in one or both ears.

RITCHIE RODGER said he had made a few tentative experiments with regard to the pitch range of the human voice, which he mentioned in a paper many years ago. Other observers had placed the average range of the speaking voice higher than he had found it at that time, but he still thought the ordinary range of the voices heard at meetings was between 200 and 250 double vibrations per second. He did not know whether Mr. Gilhespy could explain the vowel and consonant sounds being so much higher in the series which he had shown previously.

SYDNEY SCOTT said he had once examined with Bezold-Edelmann series of forks a patient who could hear *nothing* above 1,000 vibrations a second by air or bone, yet was able to hear certain spoken words if the conversational voice were raised.

W. M. MOLLISON: That patient must have been educated to hear without the S and the P.

1 *Proceedings*, 1932, xxv, 881 (Sect. Otol., 81).

JOINT DISCUSSION No. 7

Section of Surgery and Section of Medicine

Chairman—GEORGE E. GASK (President of the Section of Surgery)

[*March* 7, 1934]

DISCUSSION ON HÆMATEMESIS

Dr. Adolphe Abrahams: The most common cause of hæmatemesis is an acute or chronic peptic ulcer, and in this connexion one may include those instances of hæmatemesis which occur without any previous history of indigestion or with the irregular dyspepsia of the kind attributable to chronic appendicitis when an acute ulcer is also responsible. The frequency of hæmorrhage from peptic ulcer is substantially below that originally accepted. Of gastric ulcers about 20% exhibit hæmatemesis or melæna or both—25% in the case of duodenal ulcer—so that hæmorrhage is a familiar but far from pathognomonic symptom.

Other gastric causes include carcinoma of the stomach and rarely occurring innocent tumours, such as papillomata, myomata and angeiomata. Whilst some degree of gastric bleeding is universal in a malignant growth of the stomach, a frank hæmatemesis is comparatively infrequent; Balfour, of the Mayo Clinic, states that it occurred in 8% of the cases that underwent operation.

Hæmatemesis is a common and early symptom of cirrhosis of the liver; it is one of the cardinal features of splenic anæmia and the stomach is one of the commonest situations for hæmorrhage in the conditions which are conveniently comprised as the hæmorrhagic diathesis. Beyond mentioning the possibility of an aneurysmal leak into the œsophagus, other excessively rare causes for hæmatemesis may be ignored as less appropriate to a discussion of this character than to a compilation by a medical student of a list of causes for examination purposes.

The recognition of hæmatemesis rarely offers any difficulty. Blood from the nose or lungs sometimes presents a temporary uncertainty. Occasionally, whether as the result of an honest mistake or of deliberate deceit, the material vomited may be misinterpreted. But in any instance of gross hæmatemesis, quite apart from the accompanying anæmia blood in the stools will always be forthcoming, either as melæna or as occult blood, even if no other evidence is available.

If I have rightly estimated the primary purpose of this discussion, it is to distinguish those examples of hæmatemesis which are to be regarded as one of the unappropriated blessings of the physician from those which are the perquisite of the surgeon. To that extent differential diagnosis of the causation of the hæmatemesis is pertinent, but having regard to the character of the conditions which are usually encountered and which I have almost perfunctorily enumerated, any further details would be superfluous. I feel that I can best avail myself of the privilege of introducing this subject by suggesting the lines along which the subsequent discussion might run, and leaving to those more experienced than I the task of dealing at length with the details. My colleague, Dr. Izod Bennett, will welcome the opportunity to consider the treatment popularly termed " medical," which might more suitably be described as " non-surgical."

It is pertinent to recall that in 1924 the Sections of Surgery, Medicine, and Pharmacology and Therapeutics combined to discuss an important part of our present subject under a somewhat different heading[1] and it is interesting to observe what differences of opinion then existed not only between surgeons and physicians but between surgeons or physicians *inter se*. What change in thought and opinion have the last ten years induced in regard to the treatment of hæmorrhage from peptic ulcer, the subject of the symposium in 1924, and the predominating element in our discussion to-night?

Advances in hæmatology have had their influence in improving technique in blood transfusions and, as a consequence, in extending its therapeutic range. The more efficient administration of iron salts is now applied to the secondary anæmia of hæmatemesis, as in the microcytic anæmia of Witts for which they are so eminently suitable. If I may interpose a personal opinion, I would say that we have learned to accept with comparative equanimity a degree of anæmia which at one time would have induced panic and an irresistible impulse towards some urgent and desperate life-saving measure. I recall the dictum that life was impossible with a hæmoglobin level below 25%. I should be sorry indeed to place on an experimental basis a determination of the lowest hæmoglobin value with which existence is just possible, but it is a very familiar experience to see values below 20% in patients whose adjustment to this level is tolerably comfortable, and whose attachment to life, if precarious, is still at least sufficiently evident to deliver us from a terror that they are beyond aid if any further bleeding should occur.

To-night, as in 1924, I do not doubt that the chief discussion must refer to the comparative risks to life of hæmatemesis and of operations upon exsanguinated patients during or soon after hæmorrhage. It is evident from the contemplation of published figures that considerable difference of opinion still exists as to these mortality rates. Conybeare's investigation of the Guy's Hospital records from 1911 to 1920 revealed 23 deaths from hæmatemesis in 600 cases. Stewart's statistics for 9,000 consecutive autopsies gave an almost similar result, leading to the conclusion that the mortality from severe gastric hæmorrhage is 2·5%.

Bulmer's figures are, however, less reassuring to the physician. His investigations comprised the experience of one large provincial hospital, extending over thirty years, and of the 578 patients admitted for severe hæmatemesis from peptic ulcer during that period, more than 10% died, the death-rate among men being at least double that among women.

Our surgical colleagues will be prepared with figures with which to compare the preceding, and our combined efforts should naturally be directed to the identification of such sufferers as would inevitably die if left without operation, a certain number of whom surgical intervention would assuredly save. It has been repeatedly pointed out that the patients who die are those who manifest continued or recurrent

[1] *Proceedings*, 1924, xvii, " Discussion on the Treatment of Severe Gastric and Duodenal Hæmorrhage " (Sects. Surg., Med., and Therap. and Pharm.).

bleeding and that the single large hæmorrhage rarely kills; that the older the patient the higher is the mortality; and that males are in greater danger than females. With these reflections before us, we may establish it as an axiom that an indication for operation during hæmorrhage is the occurrence of bleeding from a chronic ulcer in a patient whose arteries are so degenerate that they are unlikely to contract sufficiently for satisfactory plugging by thrombosis.

Yet I do not feel convinced that the position can be summed up so simply as that. Are there not other acute lesions in which hæmorrhage cannot be stayed by so-called medical measures and in which some sort of operative procedure must be contemplated? It may be replied that when laparotomy has been performed in such cases no localized lesion could be identified permitting of effective treatment. With such experiences in their memories, surgeons probably still prefer to let these patients take their chance albeit a desperate one: and yet since a certain number of these patients inevitably die, one asks if recognition of this special group encourages any new surgical approach.

Apart from surgical emergencies we shall also feel called upon to debate the advantage of operation to obviate future bleeding.

Undoubtedly certain individuals with ulcer appear to have a special tendency to bleed and in this respect a familial element has been identified. Women too tend to bleed more readily than men, a factor which may influence decision. Hurst maintains that the liability to hæmorrhage is just as great after an operation has been performed for peptic ulcer as after medical treatment, whether the patient has already had hæmorrhage or not. In his experience, neither the occurrence of hæmorrhage in the past nor the danger of hæmorrhage in the future in a patient with an ulcer which has not bled, can be regarded as in any way pointing to the desirability of surgical rather than medical treatment. With this, I anticipate, agreement will be far from universal. *Inter alia*, it will perhaps be contended that the unsuccessful operations planned to obviate future hæmorrhage were wrongly planned, a circumstance which does not animadvert against surgery as a principle. Dr. Robert Hutchison, the Chairman of the 1924 symposium, expressed his belief that if a discussion on this subject were held ten years later it would be found that practice had swung more and more in the direction of those surgeons who favoured attack upon the bleeding point.

Apart from peptic ulceration, surgery has no place in the treatment of other causes of hæmatemesis, except when splenectomy is advisable, and the value of this procedure in splenic anæmia and the hæmorrhagic diathesis is almost beyond debate. Yet I anticipate a certain difference of opinion as regards the indications, more especially if other examples are forthcoming to supplement the very striking pair described by Wood and Gideon. Their patients were young men, aged 30 and 32 respectively, suffering from splenic anæmia for which splenectomy was performed. In one instance hæmatemesis did not occur at all until three and a quarter years after splenectomy, following which death took place seven months later. In both instances temporary improvement followed the operation but, in both, subsequent hæmatemeses were frequent and profuse. At autopsy, multilobular hepatic cirrhosis was found with large varicose veins round the lower part of the œsophagus.

If such examples can be multiplied it would be open to doubt whether splenectomy can prevent or retard cirrhotic changes in the liver; and search after the particular indications for this operation in splenic anæmia would be urgently indicated.

Whilst pyrexia cannot be regarded as the usual expectation during convalescence from hæmatemesis, a certain degree of otherwise unexplained fever occurs sufficiently frequently to admit of a direct association and to be accepted as demanding no particularly anxious search for a more obscure cause. The most obvious explanation is the presence of blood-clot in the colon, with bacterial decomposition and the

absorption of products of putrefaction. An alternative explanation is the infection of the ulcer itself, in support of which it has been advanced that pyrexia sometimes precedes the actual hæmorrhage. I suggest that the obvious is the more probable although the second explanation is not to be summarily dismissed. It is at least peculiar that, if stagnating blood-clot is responsible, pyrexia is not more frequent, or even universal, in hæmatemesis. In my experience, examples of protracted pyrexia are more often encountered in women. I have three almost identical instances in one ward at the present time, yet beyond their sex there is no apparent common factor and nothing distinguishing them from cases of hæmatemesis which are apyrexial throughout.

There is one special feature of ophthalmic interest relating to gastric hæmorrhage. Whilst blindness may follow any severe hæmorrhage—presumably from insufficient blood-supply to the nerve centres and retina—complete restoration of sight is almost invariable. But, hæmatemesis shares with hæmorrhage from the uterus a peculiar and more serious importance, in that on rare occasions amaurosis—which may not develop until a fortnight after the hæmorrhage—may be not only complete but permanent. Leber explains this as due to extravasation of blood at the base of the skull and into the sheath of the optic nerve, but what connexion can be traced between hæmatemesis and such an occurrence? Neuritis is a more probable explanation, and hydræmia as responsible for the neuritis, since as unilateral cases have been described the primary lesion must be peripheral. Pergens has collected 50 cases, in 86% of which the patients remained permanently blind. He says that the sex incidence is two to one in favour of males. I myself have encountered two examples in elderly women. It is clear that whatever the explanation, extensive hæmorrhage *per se* cannot be exclusively indicted or more examples would certainly be described especially by military surgeons. And although there is, admittedly, some relation to the stomach, it is evident, having regard to the rarity of the lesion after hæmatemesis, that there must be some pre-existing factor or condition of supreme ætiological importance.

Mr. G. Gordon-Taylor.—No clinical mishap is more dramatic, and none is more productive of alarm to the patient and consternation to the onlooker, than is the vomiting of blood; nor perhaps has any phenomenon a more varied or multifarious pathology. Although in a surgeon's mind ulcer-hæmorrhage may be foremost of the long list of conditions that are associated with hæmatemesis, a less hasty and more discriminating contemplation will remind him of the formidable collection of morbid states of which this symptom may be the most conspicuous feature.

Discrepancy of statistics.—Statistics concerning the relative frequency of the various pathological states associated causally with hæmatemesis exhibit much diversity in different countries, and even in the experience of authors in the same country; they can vary widely in different institutions in the same town: there appear also to be divergencies of opinion according as the figures are culled from the records of medical or surgical workers. Thus Pauchet [1], of Paris, affirms that 90% of the cases of profuse hæmatemesis concern gastroduodenal ulceration, and that only 10% of the hæmorrhages are due to hepatic, splenic, or appendix disease; from the same city, however, emanate the findings of Gutman and Demole [2], who, utilizing the statistics of the Salpêtrière, found that gastric and duodenal ulcer is the cause of only 19% of the cases of hæmatemesis investigated.

Gastroduodenal ulcer	19%	Appendicitis	5%
Cholecystitis (especially non-calculus cholecystitis)	15%	Cancer of stomach	3%
Hepatic disease	6%	Disease of spleen, High blood-pressure ...	are other causes
Chronic gastritis	6%	Causes unknown	21%

While Delore [3] of Lyons affirms that gastroduodenal ulcer is responsible for half the cases of hæmatemesis, Finsterer [4] of Vienna thinks that out of every hundred grave hæmorrhages from the stomach, in 98 the source is some large artery in the gastric or duodenal wall, and that in only 2% can the liver or spleen be inculpated.

In this country Charles Miller [5] has given an exhaustive series of conditions which may produce hæmatemesis. Apart from ulcer of the stomach and duodenum, there are included such pathological states and causes as trauma, corrosive poisons, mitral stenosis, endocarditis, miliary tuberculosis, cirrhosis of the liver, syphilitic disease of the liver—possibly associated with syphilitic gastritis—hyperpiesia, cancer of the stomach, and gastrostaxis. To this list he has added some causes of greater or smaller surgical significance, such as cholecystitis, a fibrous deformed appendix, the gastric type of visceroptosis, the "cesspool colon," and certain diseases of the spleen.

Even this does not complete the morbid catalogue, for there are still conditions which may exceptionally determine hæmatemesis. Jonathan Hutchinson, Junr. [6], drew attention in 1910 to those rare cases of gastric hæmorrhage of septic origin. Whether these complicate the post-operative course of some surgical intervention or whether they originate apart from surgery, they are of grave prognostic significance, and their appearance is usually, if not invariably, the harbinger of the approaching end. Such cases are hardly likely to be considered as amenable to surgical intervention.

With the exception of the ulcer-hæmorrhages, there can scarcely be an occasion when urgent surgery may legitimately be discussed as a method of hæmostasis, and the oppressive burden of pathology recorded in the foregoing paragraphs must give the would-be operator cause to think again and yet again before deciding to intervene surgically during acute hæmorrhage from the stomach.

Whether surgery "*à froid*" is the appropriate measure for a particular case of hæmorrhage due to a gastric carcinoma can only be determined after radiological examination, and in most cases the possibility of removal—the only measure of real value—can only be settled after laparotomy; the hæmorrhages of splenic anæmia are not always permanently arrested by the removal of the spleen, and the operation, which may require considerable technical skill, demands also some forethought, perhaps preliminary radiation, and the employment of expert hæmatology, not excepting a minute enumeration of blood-platelets.

In these cases, and in diseases of the gall-bladder or appendix, and in cases of essential thrombocytopenia, surgery performed at the time of election may indeed play a useful and curative part, but in most of the other conditions associated with hæmatemesis which have been mentioned, operative surgery can belong only to the ignorant, the commercial, or to the experimentalist, and will naturally be dismissed from further consideration by any surgeon of judgment.

Before any decision is taken on the matter of operating for, and in the presence of, a severe hæmatemesis, the greatest caution must be exercised to ensure absolute accuracy in the diagnosis of the causation of the hæmorrhage. There are, as has been shown above, many conditions which produce hæmatemesis, but the only pathological state which justifies urgent surgery is that of *bleeding from a chronic ulcer*. There should preferably be clinical, radiological—even biochemical—evidence of the existence of a chronic ulcer, or if the details of the patient's previous history are not available, the anamnesis should leave in the observer's mind no doubt as to the existence of a chronic ulcer of stomach or duodenum, and no uncertainty as to this being the source of the cataclysmic menace to the patient's safety. Nothing that fails to conform to this standard should be regarded as a pretext or as an indication for operating; for surgery misdirected can only jeopardize most grievously the patient's life.

Papin and Wilmoth [7] insist that though timely surgery may alone be capable, on occasion, of saving certain lives, it demands a combination of the highest clinical judgment and no inconsiderable technical competence.

The role of surgery in hæmorrhage of ulcer-origin.—Is surgery to be employed as a prophylactic measure against the hæmorrhage of gastroduodenal ulceration?

An answer to this question would appear to depend upon: (*a*) The incidence of hæmorrhage in connexion with ulcer; (*b*) the risk to life with which ulcer-hæmorrhage is fraught, and (*c*) the prospective mortality of any surgical measure contemplated.

(a) *The incidence of hæmorrhage*: A history of hæmatemesis or melæna or both was obtained by Hurst [8] in 18% of the cases of chronic gastric ulcer and in 25% of the duodenal ulcers examined at New Lodge. The statistics of Lord Moynihan [9] and of Balfour [10] of the Mayo Clinic are similar; 20% of gastric ulcers and 25% of duodenal ulcers have bled.

Crohn and Manheim [11] of the Mount Sinai Hospital, New York, found evidence of bleeding in nearly 30% of gastric ulcers and in nearly 20% of duodenal ulcers admitted; they insist, however, that the percentage of ulcer-cases gaining admission to hospital is relatively small, and that probably only 10% of all ulcers bleed.

The cases admitted to New Lodge are also necessarily of a more severe type, and Hurst's estimate [8] of the percentage of ulcers that bleed is also about 10%, which agrees with the figures of Conybeare [12] computed from the statistics of Guy's Hospital.

(b) *What is the risk to life from ulcer-hæmorrhage?* Conybeare [12] affirms that only 2% of patients with ulcers that bleed die of hæmorrhage; so that the risk of loss of life by bleeding from an ulcer is only about 1 in 400 or thereabouts. On the other hand, Ernest Bulmer [13], of Birmingham, found that out of a total of 249 cases of hæmatemesis due to chronic peptic ulcer, death occurred in 29—a mortality of 11·6%: 89 of his cases were males, and of these 15 died—a mortality rate of 16·8%. In an editorial in the *British Medical Journal* at the end of 1929 [14] the ominous statement is made that in 20% of ulcer cases the patients die of bleeding if not treated surgically. Although the extravagant claims made for the safety of the medical treatment in peptic ulcer may not be substantiated, *the chances of any patient with ulcer dying of hæmorrhage should not be more than 1 in 50*, but this risk will naturally increase with the length of the history and the chronicity of the ulcer. The decision in favour of surgery in peptic ulcer is not likely, however, to be determined on the question of prophylaxis against hæmorrhage. In the case of gastric ulcer, chronicity, the menace of malignancy, severity of pain, incapacity for work, etc., are more likely to be the deciding factors.

There is at the moment a bias in favour of an expectant attitude in the case of a duodenal ulcer. Many are "abstentionists," regarding as indications for operation only stenosis, a large crater, duration of existence, perforation, and hæmorrhage. The only operation which is going to confer an immunity from hæmorrhage is a resection, and in the case of duodenal ulcer I feel inclined to waive any plea for a preventive operation of this character until a time when the patient may genuinely be said to have earned his operation.

Does a history of hæmorrhage constitute an indication for surgery in chronic peptic ulcer? The authoritative opinion of D. P. D. Wilkie [15] favours medical treatment for the first hæmorrhage, and in the event of a second bleeding it is recommended to wait for ten days after it has ceased and then to operate. It is admitted, however, that three patients died *during* the second hæmatemesis.

The mortality of cases of hæmatemesis from chronic ulcer of stomach or duodenum treated medically at the Middlesex Hospital from 1924-1933 inclusive, and in which a second large hæmorrhage occurred, was no less than 78%, and with each successive bleeding the death-rate mounts higher. This seems to indicate the

need of surgery before a second hæmatemesis occurs. Even Bulmer [13] avers that in the event of hæmatemesis being followed by the presence of occult blood in the stools for some weeks, surgical interference should be utilized without delay; this opinion coincides with the view of Charles Bolton [16] based upon a wealth of experience which makes it of supreme value.

Does active hæmorrhage from a chronic peptic ulcer, whose existence is beyond cavil, require surgical treatment during the bleeding, or is surgery best practised after the bleeding has ceased?

It is doubtless more convenient, and less harassing and disturbing, to operate with deliberation at the time of election after the first bleeding has ceased. But how do we know that it will cease and when it will cease?

Dr. Robert Hutchison [17] when summing up the debate on the treatment of severe gastric and duodenal hæmorrhage in 1924, stated that in his own practice he had followed the teaching of Mr. Sherren and did not advise operation while bleeding was persisting, but *recommended interference not later than forty-eight hours after it had ceased.* Hutchison had several times repented having waited longer than forty-eight hours, for he could recall several cases in which the patients had died through delay in operating. He opined that in ten years from 1924 the tendency of treatment in grave *hæmorrhage would be towards a direct attack while bleeding was going on.*

Is urgent surgery ever required in connexion with active hæmatemesis?

Hans Finsterer [4] regards bleeding from a chronic ulcer as an imperative indication for immediate surgery. With most surgeons it is the repetition of bleeding that demands operative interference, but Finsterer regards hæmatemesis as almost as urgent as a perforation.

The failure of surgery "*à chaud*" in the hands of other operators (Savariaud had an operative mortality of 62%, Tuffier 36%) has been the result of late interference in cases treated unsuccessfully by medical measures. In the experience of the Viennese surgeon, operation must be performed within forty-eight hours, and preferably within the first twenty-four hours; therefore the third day is the critical day, and the mortality goes up enormously after this. Finsterer's figures present an extraordinary testimony to the safety of this policy in his hands.

IMMEDIATE OPERATION (24-48 HOURS). (FINSTERER).

	No. of cases	Deaths	Mortality rate
Gastro-jejunostomy ...	7	1	14·2%
Gastrectomy	39	1	2·5%
	46	2	4·3%

LATE OPERATIONS (PRELIMINARY MEDICAL TREATMENT). (FINSTERER).

	No. of cases	Deaths	Mortality rate
Gastro-jejunostomy ...	7	3	43%
Excision of ulcer	2	1	50%
Gastrectomy	46	14	30·4%
	55	18	32·7%

Gur, following the same plan of early operation, had a mortality rate of 11%, and Bohmanson of 20%.

My own attitude towards these cases is divisible into four periods. (*a*) A period from 1919 to 1924, when I was still profoundly influenced by the dramatic results of blood transfusion, to which I had been introduced during the Great War, and of which I had had considerable experience in France and Flanders. During this period of active surgical treatment of acute hæmatemesis, 20 out of 22 cases recovered from operation—a mortality of 9% (18).

(*b*) The period 1924 to 1926, during which operation was performed in only 10 cases of acute hæmatemesis, with four deaths, i.e. a mortality rate of 40%. The

high mortality rate may have been due to the fact that many were dealt with in institutions remote from personal supervision, and without all the arrangements necessary for adequate and repeated transfusion. A spirit of self-criticism and a disinclination to operate *à chaud* were thereby induced. Nevertheless, the total in groups (*a*) and (*b*) amounted to 32 cases, with six deaths—a 19% mortality rate.

(*c*) A period of abstention from active surgery in these cases; this attitude coincided with the impetus given to the medicinal or alkaline treatment of ulcer, resulting from the papers of MacLean, and the booming of this ancient method of therapy in the lay press. During this period, the mortality of acute hæmatemesis from chronic ulcer which I saw in outlying hospitals and in private practice was enormous; there were no operations for active hæmatemesis at the Middlesex Hospital during this quinquennium.

(*d*) A period of renewed surgical interest in these cases, in which it is hoped that some information for guidance in the selection of the right case for operation *à chaud* may be accumulated,—the period of "opportunism."

SURGICAL TREATMENT OF ACTIVE HÆMATEMESIS. (GORDON-TAYLOR).

	Cases operated	Mortality	Percentage of mortality
(*a*) 1919-1924 ...	22 ...	2 ...	9%
(*b*) 1924-1926 ...	10 ...	4 ...	40%
	32 ...	6 ...	18·75%

(*c*) 1926 till recently: a policy of abstention with almost universally fatal results.

An investigation has been made for me by three of my assistants at the Middlesex Hospital—Roy Banham, Alan Kekwick, and J. Knights—of the results of *medical treatment of the cases of hæmatemesis* from ulcer, or supposed ulcer, admitted into the wards of the Middlesex Hospital from 1924 to 1933.

Total of cases of hæmatemesis admitted	124	
Cases showing positive X-ray evidence of ulcer a few weeks following hæmatemesis	47	Total proved ulcer 67
Deaths from hæmorrhage	20	

Four of these cases of fatal issue have been excluded in computing the percentage of mortality, as the patients were completely beyond mortal aid. In many of the fatal cases, too little use may have been made of blood transfusion, and with increased employment of this form of therapy, some patients might have been saved from death. In the opinion of those who collated and analysed the cases for me, the condition of the ulcer at the time of death was such that for only about 12 out of the 16 patients could anything have been attempted in the form of surgery. The post-mortem room, however, shows the end-result of a process that might have been prevented or checked by timely surgery.

Of the patients who died, one was found to have, in addition, a new growth of one of the bronchi; a second died a few minutes after admission, from a gastro-jejunal ulcer, and a third had a fairly extensive cancer of the lesser curvature of the stomach; a fourth died of hæmatemesis associated with a perforative peritonitis. There are therefore only 16 deaths in which surgery might have triumphed over medical methods, i.e. a mortality rate of 24%.

There are, however, certain other facts that have been elicited by the inquiry.

(1) The average duration of life in hospital after the primary hæmatemesis in cases ending fatally	9·9 days
(2) The number of cases who died of hæmatemesis *while undergoing intensive medical treatment for ulcer*	6 ,,
(3) The fatality of a second hæmatemesis was 78%, and the risk to life increased with such bleeding.	

Could surgery have aided those cases in the Middlesex series which ended fatally?
Appended is a list of the post-mortem findings in the fatal cases in the Middlesex series.

CASES OF FATAL HÆMATEMESIS ADMITTED INTO MIDDLESEX HOSPITAL, 1924-1933, WHO WERE TREATED BY NON-OPERATIVE MEASURES AND ON WHOM AN AUTOPSY WAS PERFORMED.

	Sex	Age	If diagnosed as ulcer before hæmatemesis	Cause of death	Duration of life after hæmatemesis	Lesion found at post-mortem	Contra-indication to surgery
1	F.	50	Yes	Cardiac failure after recurrent hæmatemesis	10 days	Pyloric ulcer	Extensive lesser curve carcinoma
2	F.	53	Yes	Cardiac failure after recurrent hæmatemesis	14 ,,	G. U.	—
3	M.	73	No	Recurrent hæmatemesis	4 ,,	Posterior wall ulcer	Coexistent carcinoma of bronchus
4	M.	45	Yes	Recurrent hæmatemesis	7 ,,	Posterior wall ulcer eroding pancreas	—
5	F.	54	Yes	Cardiac failure	7 ,,	Lesser curve ulcer	—
6	M.	45	Yes	Cardiac failure and anæmia	7 ,,	Lesser and greater curve ulcers	—
7	F.	62	Yes	Primary hæmatemesis in hospital	10 mins.	Gastro-jejunal ulcer	Death almost immediately after admission
8	M.	59	Yes	Cardiac failure and anæmia	1 day	D. U. adherent to pancreas	—
9	M.	51	Yes	Primary hæmatemesis in hospital. Cardiac failure	1 ,,	Lesser curve ulcer	—
10	M.	35	No	Pneumonia	21 days	D. U. attached to gall-bladder and pancreas	—
11	M.	65	No	Bronchopneumonia	14 ,,	Small lesser curve ulcer	—
12	M.	48	Yes	Anæmia. Primary hæmatemesis in hospital	2 ,,	D. U. adherent to pancreas	—
13	F.	57	Yes	Perforation followed by hæmatemesis. Peritonitis	3 ,,	D.U.	Diffuse perforative peritonitis
14	M.	56	Yes	Primary hæmatemesis in hospital. Cardiac failure	12 ,,	Pyloric ulcer	—
15	F.	62	Yes	Primary hæmatemesis in hospital. Cardiac failure	13 ,,	Pyloric ulcer	—
16	F.	49	No	Cardiac failure and anæmia	5 ,,	High greater curve ulcer	—

There is, of course, no one type of operation which is appropriate to every case; the object of operation in these cases is to stop hæmorrhage, and in my own first series this included such a variety of operative measures as gastrotomy with ligature of the bleeding vessel, pyloric occlusion and gastro-jejunostomy, cautery and gastro-jejunostomy, sleeve resection and gastrectomy.

Are there any clear indications for urgent surgery for those surgeons who are "opportunists" or "interventionists"?

(1) If there is pyloric or duodenal stenosis, operation alone may save life in the presence of hæmatemesis.

(2) Certain degrees of mid-gastric narrowing.

(3) The existence of a large or deep chronic ulcer, especially if the history is a protracted one, or the ulcer of penetrating variety.

(4) Super-added perforation.

Suggestions for surgeons operating for urgent hæmatemesis.

(1) Make decision *at once*: delay increases risk of operation.

(2) There must be possibilities of unlimited blood for transfusion.

(3) Operate as far as possible under local anæsthesia, reducing strength of solutions; in the event of extreme gravity of the patient's condition, dispense with splanchnic anæsthesia. General anæsthesia to be the lightest possible.

(4) If there is much blood in the intestine, perform cæcostomy and practise lavage of the large bowel.

Hæmatemesis and perforation.—There is one group of cases to which a brief reference may be made. In a somewhat insignificant minority of ulcer hæmorrhages there may be a concomitant perforation. The association is fortunately rare, for there is much truth in the dictum of Moses Behrend [19]: "Perforated ulcers bleed rarely; bleeding ulcers perforate only very exceptionally."

The two complications of a peptic ulcer may occur simultaneously and perforation will, of course, demand urgent operation, or the twin nature of the catastrophe may be discovered only during the course of the operation. The association of an active hæmorrhage will naturally call for some modification of the technique habitually employed in dealing with perforation of a peptic ulcer.

I have actively intervened for this syndrome, three times in connexion with gastric ulceration, and once for a bleeding and perforated duodenal ulcer. All four cases were dealt with by resection, and all four patients recovered. I have, however, dealt unsuccessfully with a perforated jejunal ulcer consequent upon a gastrectomy for duodenal ulcer; the jejunal ulcer was actively bleeding at the same time. In another case, after an exploration, I found it impossible to do anything for a patient with a bleeding gastro-jejunal ulcer, which still further complicated the clinical picture by eroding the transverse colon. In this last-mentioned case with its ghastly communications, a bloody flux composed of diarrhœa and putrid melæna poured through the pathological anus into the stomach, whence it was rapidly ejected; the victim's sufferings were fortunately ended by death.

Hæmorrhage from a peptic ulcer may *precede* perforation; the fatality of this combination and sequence of complications is prodigious. In these cases the enfeebled condition of the patient resulting from the hæmorrhage may mask the ordinary clinical picture of a perforation which may fail to be diagnosed.

Finally, hæmorrhage may complicate the progress of a successful operation for perforation. In my own experience the results have been calamitous, whether the bleeding has been attacked by surgical re-intervention or treated by medical measures. These hæmorrhages are usually ushered in by an increased pulse-rate and some elevation of temperature; they occur, as a rule, from seven to twenty-one days after the initial emergency operation. The hæmorrhage may originate from the sutured ulcer itself, from some other ulcer in the stomach, or it may be a diffuse parenchymatous bleeding from the gastric wall. In two cases of my own, this last-mentioned condition of the gastric mucosa was found, and both these ended fatally.

Is there any prophylactic measure against this most lethal combination of catastrophe? Resection of stomach or duodenum can hardly be urged as a routine measure for perforated ulcer, though Alessandri [20] speaks well of it in early cases and, amongst others, Julian Taylor [21] has employed it in perforations of stomach-ulcers. In the hands of skilled and experienced surgeons it may be a sound measure, but an enormously increased mortality from perforated ulcer would result if this operation were regarded as the appropriate form of surgery in every case.

Post-operative hæmatemesis.—This complication of gastric operations due to failure to occlude bleeding vessels in the stomach or intestinal wall is all but unknown to me, since I rarely, if ever, employ clamps. Indirect operations for peptic ulcer of stomach or duodenum may lead to hæmorrhage from the original

ulcer, which is preferably dealt with by excision or exclusion. A fatal bleeding may take place from such a source, unless surgical re-intervention is employed.

Anastomotic ulcers.—If these are complicated by severe hæmorrhage, the rules governing the treatment of peptic ulcers of stomach and duodenum should control the surgeon in this class of case. Amidst the anxieties of a complex artificial pathology, if urgent operation should be required or undertaken, the least possible should be done at the time; hæmostasis alone is to be secured.

In conclusion, my post-war attitude towards these cases of ulcer-hæmorrhage has been first, that of an "interventionist," and then that of an "abstentionist." I have of late made trial of medical measures in cases of hæmatemesis of ulcer-origin, but have been disappointed by the appalling results of this policy of abstention, in the relatively limited number of cases with which I have been brought in contact. Sporadic cases of surgery practised during this period of abstention, in cases of acute hæmorrhage, have on several occasions ended successfully, but apart from these beacon-lights showing the way, the mortality of the non-operated cases has been almost 100%.

However, I am taking comfort in the reflection that this series of cases with which I have been in recent years concerned must have been of a peculiarly grave character, and that I must have been as unlucky in my clinical material as I have been unfortunate in the treatment, or rather the lack of treatment, that I have advised. These results of mine are in contrast with the Middlesex Hospital figures relating to the non-operative treatment of hæmatemesis. The Middlesex Hospital mortality in these medically treated cases, amounting to 24%, is almost identical with the figures collected by A. M. Cooke [22] from 191 cases of gastroduodenal hæmorrhage admitted into St. Thomas's Hospital during seven years. In 24·1% of these, the patients died.

These two sets of figures surely show that the medical treatment of active bleeding from a chronic ulcer is still capable of much improvement. I would plead for closer co-operation between physician and surgeon in the conduct of these most anxious cases, so that some agreement may be attained as to which cases demand an expectant line of treatment and which can be saved by surgery alone. Every case has to be considered on its own merits or demerits; there can be no universal rule. Intercurrent illness, the previous habits and physical conformation of the patient, the personal courage of the patient as well as of the operator, the environment, and the technical skill of the surgeon, may all be factors which influence physician and surgeon in arriving at a decision as to the line of treatment. As in so many surgical undertakings, it is judgment that counts.

Above all, if surgery be considered with any seriousness, let there be no procrastination, for delay is indeed fraught with danger. Early enterprise is the prelude to success.

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Dr. T. Izod Bennett: Mr. Gordon-Taylor emphasized an important point when he said that the cases in which surgical aid should be sought, or consultation between physicians and surgeons immediately arranged, are essentially those in which we have clear indication that the bleeding is coming from a chronic ulcer, and especially those in which there is a stenosis either of the pylorus or in the duodenum. In actual fact, however, one finds that these form a small proportion only of all the cases of hæmatemesis to which one is called. The figures which Mr. Gordon-Taylor showed are in some ways deplorable, but if we are to keep our sense of proportion in such a discussion as this we must, at the outset, remember that the cases of hæmatemesis admitted to any general hospital constitute a select and special class. I would ask my surgeon-colleagues to give careful consideration to this suggestion—that by far the greater number of those cases should never have been admitted to hospital at all. There are few types of emergency with immediate threat to life in which the consulting physician is called in with greater frequency than in those of severe intestinal hæmorrhage. Hæmatemesis and severe melæna, with collapse, constitute a large proportion of the emergencies to which I am called in consultation. I do not see these cases in hospital, I see them mostly in the London suburbs.

The first thing I say is "On no condition must this patient be moved." I do not say it as a plea for medical rather than surgical treatment; I say it in order to emphasize the fact that patients who are admitted to a hospital on account of hæmatemesis are brought there either through mistaken judgment or because the home circumstances were such that it was impossible to look after them outside hospital. There can be no more difficult decision than to move a patient whose life is in danger owing to bleeding from some intestinal lesion. For the same reason the emergencies by which a surgeon is confronted with the question of operation or not, are essentially of the worst prognosis. And that is why it is unfair and misleading to quote statistics as to the mortality following operation for hæmatemesis and the mortality after medical treatment. It is obvious that the surgeon will not be called in if the case appears to be one of a mild hæmatemesis with a likelihood of recovery, therefore it is absurd to quote results of surgical, as opposed to medical, treatment in those cases. But, with those reservations—and they are serious reservations—I agree with Mr. Gordon-Taylor that in cases of hæmatemesis with obvious risk to life, whenever there is an obvious history of previous gastric ulcer, or where there is obviously pyloric or duodenal stenosis, there should be close consultation between physician and surgeon, possibly with a bias in favour of surgery.

In my own experience I can trace seven deaths in the last ten years in cases of hæmatemesis. In all of them the ultimate diagnosis was known. One of them was an example of a rarity to which Dr. Abrahams has referred—a case in which an aneurysm had ruptured into the duodenum. Two were cases of marginal ulcer following the operation of gastro-enterostomy many years previously; one was a case of Banti's disease, one a case of cirrhosis of the liver; one was a case of cerebral thrombosis following a severe hæmatemesis, which would clearly have had a fatal issue in spite of treatment and did so end three days afterwards, and in the last the patient would possibly have been saved if surgery had been employed early. But when I set this instance in which the patient might have been saved by

surgical experience and skill, against the 200 or 300 cases of hæmatemesis which I have seen in that ten-year period, I feel, naturally, that the indications for surgery are exceedingly rare.

Next I should like to say what I consider the proper method of treating these cases medically. It is not fair to assume, when one sees a series of deaths after non-operative treatment, that these cases have been correctly treated. Any case of hæmatemesis should be regarded as a grave medical emergency, and should be treated with the same meticulous care as that with which every surgeon treats a case after a severe abdominal operation. It is only after several days have passed that the physician can feel otherwise than extremely anxious about such a case, and I fear that the patients in these cases, especially if the actual amount of blood vomited has been enormous, are often treated with a lack of care which, in my opinion, amounts to levitv.

The first step in such medical treatment consists in forbidding anything to be taken by the mouth—if the condition is at all dangerous—for from thirty-six to forty-eight hours. The next step is the administration of morphia, or some drug of the narcotic group, in sufficient dosage to cause twilight sleep; not only sufficient to tide the patient quietly over the first twenty-four hours, but to keep him in a condition in which he is not only not suffering from absence of food and drink, but is unaware that these have been withheld. Such a patient requires the most careful nursing, night and day, with regular rectal administration of saline. Afterwards, first fluid is given, and then solid, in a cautious manner, with continual examination of the stools for occult blood, and observation of the pulse-rate for signs of recurrent hæmorrhage, &c.

I will not dwell further on the medical details. We are only too well aware of the atmosphere of panic and despair in the household when we are called to one of these desperate cases; on all hands there is the feeling that "we must *do* something." Certainly there is much to be done, even if it is mainly of a negative character. But it is only rarely that that "something" should be a surgical operation. Even if it should be, most surgeons and physicians, seeing a patient in bed at home where the hæmorrhage has occurred, would agree that it is better to face the risk of a recurring hæmorrhage without moving him, than to move him so as to put him into better operative circumstances.

A minor point in technique is that when we begin, cautiously, to give fluid nourishment, not milk or glucose lemonade should be given, but isotonic saline by the mouth. It is only when we are satisfied that the stomach is not filling-up owing to pyloric stenosis and that the pulse-rate does not show a recurrent hæmorrhage, that we can proceed to give foods of higher nutritive value.

Mr. John Morley: Dr. Robert Hutchison's prophecy in 1924 (that in another ten years, the practice with regard to hæmatemesis would have swung round in the direction of early surgical intervention) has not I think been entirely fulfilled by events, but it is fitting that we should consider whether any fundamental changes in the situation have arisen during the past ten years.

It appears to me that two new factors of some importance have emerged. In the first place, while no great advance has been made in the technique or the physiological principles of blood transfusion, the organization of efficient transfusion services in every large city has given us a much readier opportunity of tiding patients over the immediate danger of death from hæmorrhage. And in the second place, the great advance in the knowledge of hæmatology and in the treatment of the secondary anæmia that follows hæmorrhage, enables us more surely and rapidly to restore patients who have suffered from the less grave forms of hæmatemesis to a condition in which the surgeon is not afraid to operate.

We are all agreed that hæmorrhage from acute ulcers is better left to the physicians, not because none of these patients will die, but because most will recover, and surgical intervention will jeopardize rather than help their chance of recovery.

It is only in dealing with hæmorrhage from chronic ulcers that there is legitimate room for difference of opinion. The mortality rate in cases of chronic ulcer admitted to hospital on account of hæmatemesis is, I believe, considerably higher than has been generally admitted. Out of 249 such cases admitted to the Birmingham General Hospital, Bulmer records twenty-nine deaths—a mortality of 11·6%. Out of 330 such cases admitted to the Manchester Royal Infirmary and treated in the medical wards during the last ten years, there were forty deaths—a mortality of 12·2%. I quote these figures with some reserve, as post-mortem verification of the clinical diagnosis was not always obtained, and some cases of cirrhosis of the liver and cancer of the stomach may have been included. But if we may consider that of all cases of chronic ulcer admitted to hospital on account of hæmatemesis, the mortality rate on medical treatment is somewhere in the region of 10%, it must be admitted that the problem of reducing that mortality is one of gravity and urgency.

Can we recognize with reasonable certainty the presence of a chronic ulcer in a patient suffering from hæmatemesis? I believe that we can, if there is a long history of periodic attacks of pain, coming on some long time after the taking of food, and relieved by taking food and alkalies, but I believe also that no detail in the clinical history will tell us with any degree of certainty whether the ulcer is gastric or duodenal. The length of time that has habitually elapsed between the taking of food and the onset of pain will certainly not help us, for most gastric ulcers resemble duodenal ulcers in showing a latent period of two or three hours after food before the onset of the pain. Nor is the predominance of hæmatemesis or of melæna an infallible guide, since duodenal ulcers often cause hæmatemesis first, and occasionally a gastric ulcer gives rise to melæna alone.

X-ray examination is not to be thought of within a short time of a hæmorrhage, on account of the grave risk that it may cause the bleeding to begin afresh.

What should the surgeon do when he is confronted with a patient who is vomiting blood profusely from a chronic ulcer, or who is blanched and collapsed from a recent hæmorrhage?

I do not believe that we can answer the question by statistics, or that any rule can be formulated that will be a sound guide in every case, but in making our decision we must remember two facts. In the first place, post-mortem examinations in these cases usually show a large vessel, commonly an artery, lying open in the ulcer, and secondly we have the clinical experience that in the fatal cases the patients do not die as a rule after the first hæmorrhage, but, as in cases of secondary hæmorrhage from an artery elsewhere, they succumb to a series of severe bleedings.

I believe that one should never operate on a collapsed and exsanguinated patient. To do so, is, in my judgment, likely to prove disastrous. Nor, however, do I believe that we should resign the case to the physician with a polite intimation that if all goes well we will do something in two or three months time. If we do, as I have seen to my sorrow, the patient may die within two or three days from a further hæmorrhage.

After a really severe hæmatemesis, one should operate as soon as it is reasonably safe to do so. How can we render the patient safe for an operation in the shortest time possible? An early blood transfusion is the most urgent measure, repeated a few hours later if need arises, and supplemented by rectal, and if necessary, subcutaneous or intravenous saline. But I think a period of sleep under morphia is also essential and of course no fluid must be given by the mouth. By these

measures in twelve or twenty-fours we can often secure a remarkable improvement in the circulation. When the pulse has come down to the 80's, with a good volume, and the patient's colour has improved in proportion, I think if the hæmorrhage has been a really grave one, it is safer to operate than to wait. One is always tempted to wait a little longer, in the hope of making it safer still, but I have more than once had occasion bitterly to regret too long a delay. But let me say again, unless we can secure this favourable condition of the circulation, I believe that operation is too risky and is a counsel of despair.

If then it has been decided to operate, what should our operation be? Indirect measures, such as gastro-enterostomy alone, are far too likely to be futile. I remember a man on whom I performed a simple gastro-enterostomy many years ago for a duodenal ulcer with melæna, who continued to have recurrent hæmorrhages at intervals of six months or so for years afterwards. The only satisfactory measure is to secure the bleeding vessel, and this object is most readily attained by some form of excision of the ulcer. In the case of gastric ulcers this presents, as a rule, no difficulty, except in the ulcer very high up on the lesser curvature. Whether the operation consists of local excision of the ulcer combined with a gastro-enterostomy, or, as I usually prefer, a Schoemaker type of gastrectomy, is relatively unimportant. What matters is that the bleeding vessel should be secured.

In the case of duodenal ulcers the problem is more difficult. Anterior duodenal ulcers can be excised fairly easily, but the form of duodenal ulcer that most commonly causes fatal hæmorrhage is the large posterior ulcer, deeply invading the head of the pancreas, and eroding a considerable branch of the gastroduodenal or pancreatico-duodenal arteries. In some of these an attempt at excision may involve danger to the common bile-duct or hepatic artery, and where this is so I prefer to tie the gastroduodenal artery close to its origin from the hepatic, and often to tie the pyloric artery as well, combining these ligatures with a posterior gastroenterostomy.

I will venture to make one somewhat controversial statement on the question of choosing the right moment for surgical intervention in these cases of hæmatemesis from chronic ulcer. I hope that I shall not be suspected of advocating indiscriminate operations if I express my belief that the surgeon alone has the particular type of experience that makes it possible to decide when the patient can stand an operation. The closest possible co-operation between the physician and the surgeon is, however, needed in dealing with these cases. In supervising the repeated blood transfusions that may be necessary and the frequent hæmoglobin estimations that are certainly required, it is just as important to have the services of an experienced physician as it is to have an experienced surgeon where an acute perforation of an ulcer is concerned. Lives are undoubtedly lost in some cases by leaving these patients at a time of grave crisis in the care of hospital resident officers of inadequate experience.

The only place for surgery in the treatment of hæmorrhage from acute gastric ulcer is as a measure of prevention. I am sure that occasionally, though rarely, chronic or subacute appendicitis may give rise to hæmatemesis, presumably by small erosions of a septic nature. The hæmorrhages may be alarming and recurrent but seldom grave, and removal of the appendix at a safe interval will put a stop to them. I do not refer to the cases in which the appendix is a little thickened or adherent or fibrosed. I am entirely sceptical of the occurrence of gastric hæmorrhage due to that type of appendicitis. I allude only to cases in which the appendix is found greatly swollen, red and congested, though usually there is no history of a recent acute attack of appendicitis.

Early post-operative hæmatemesis. — Where a serious degree of early post-operative hæmatemesis occurs, assuming that the surgeon has dealt adequately with the ulcer, we can only ascribe it to a failure in surgical technique. Bleeding from

a suture-line is always due to the sutures being placed too far apart, and not sufficiently tight, so that some considerable vessel escapes their grip.

I believe that the mucous loop-stitch is particularly liable to give trouble. It was my custom at one time to use this stitch for rounding the corner in a gastro-enterostomy, but some years ago I lost a patient from post-operative hæmorrhage, and post mortem a vessel was found at one end of the anastomosis which had escaped my mucous loop-suture and had caused the bleeding. From that time I have used a running stitch to round the corner. I am aware that some surgeons, not having had just such an experience, will say that the mucous loop-stitch is safe. I can only record the mistake which cost my patient his life, and mention the effect which it had on my technique. But I will admit that much the most important point is to insert one's sutures sufficiently close together.

There is one other possible source of early post-operative hæmorrhage that should be mentioned. It is the custom of many surgeons when performing a gastro-enterostomy for duodenal ulcer, to invaginate the ulcer by a row of sero-muscular stitches placed transversely, in cases in which the ulcer is not excised. My colleague, Mr. Rayner, tells me that he has seen a case of grave post-operative hæmatemesis after this procedure. He believes—and it seems to me probable enough—that the invagination by sutures of the duodenal wall tore open the crater of the ulcer and ruptured a vessel already eroded by the ulcer. If the main purpose of such an invagination of a duodenal ulcer is as a safeguard against perforation, this can be obtained as surely by stitching omentum over the ulcer, without any risk that bleeding may be caused.

Remote post-operative hæmorrhage is usually from an anastomotic ulcer, either on the line of the anastomosis or in the efferent loop of the jejunum. These ulcers—often on the posterior margin of the stoma, and associated with great hypertrophic fibrosis—provide, even under the best conditions, a searching test for a surgeon's skill. I am never disposed to operate on such a patient until the anæmia resulting from the hæmorrhage has been cured, for I know he will need the best quality of heart and blood if he is to stand successfully the severe operative procedure that will certainly be necessary. Nor would I operate on such a patient at all if I thought there was still a reasonable chance of a cure by medical means.

Diseases of the spleen.—I have little to say on the subject of hæmatemesis associated with disease of the spleen. It is now generally recognized that in thrombocytopenic purpura nothing short of splenectomy may avail to stop the repeated hæmorrhage from the stomach and elsewhere, and also that the spleen in these cases is not necessarily enlarged. It seems probable that more frequent thrombocyte estimations and the use of the stasis test would show that some of the cases regarded as cases of acute gastric ulcer or gastrostaxis fall into this group.

In two cases of splenic anæmia associated with repeated hæmatemesis, both with a moderate reduction of the thrombocyte count, in which I performed splenectomy at the urgent behest of a physician, in spite of some improvement in the patient's health, the hæmorrhages have been repeated at intervals since the operation over a period of more than two years. I conclude that when œsophageal varices have formed in such cases splenectomy does nothing to cure them.

Dr. R. S. Aitken: The main choice in the treatment of hæmatemesis lies among three courses: (1) purely medical treatment (of which the essentials are rest and morphia), (2) medical treatment plus blood transfusion, and (3) surgical treatment. In the majority of cases the hæmorrhage is not severe enough to threaten life, and such patients are properly treated medically; they recover. The grave cases, however, constitute a problem, and when statistics and case records are discussed,

some attempt should be made to separate these from the milder type. I have made such an attempt in examining the London Hospital records of the past five years. Out of 253 cases of bleeding from stomach and duodenum, 61 may be distinguished as grave cases, on the ground of reduction of red cells to 2,000,000 or less, and of hæmoglobin to 20% (Dare) or less, or on clinical evidence of a similar degree of exsanguination. Of these 61 patients, 25 died; the mortality was heaviest among those treated on purely medical lines, least among those treated medically and transfused, and intermediate in the group in which the abdomen was opened. The figures are so few, and the varying circumstances and features of different cases are so many, that no final conclusions in a scientific sense are justifiable. But two definite impressions are conveyed: first, that the actual mortality among cases treated medically is by no means negligible, and second, that the best course in the management of cases of grave bleeding is to treat them on accepted medical lines and transfuse once or oftener, resorting to surgery only if repeated transfusion fails to initiate recovery. This is substantially the attitude to the problem which Hurst adopted in 1928 in his book on "Gastric and Duodenal Ulcer."

Professor C. A. Pannett: It is eloquent of the difficulties surrounding this subject that none of the openers of the discussion have definitely decided whether, when a chronic gastric ulcer is bleeding, the treatment should be medical or surgical. I believe that is partly because the statistics, as several speakers have already said, are concerned with series which are not comparable. They are compiled so differently that one set of figures points one way, and another indicates a contrary policy.

What is particularly noteworthy is the fact that the hæmorrhages are secondary, i.e. hæmorrhages likely to be repeated. I differ absolutely from those physicians and surgeons who think that surgery should be reserved for cases with repeated hæmorrhages and that only when patients have become worse and worse, and their hæmoglobin has been reduced to 20%—i.e. when they have lost 80% of their hæmoglobin—should surgery be resorted to. These are the cases in which surgery should be absolutely excluded from the treatment. I feel that when it is known that a man has a gastric ulcer, surgery should be considered from the beginning of a hæmatemesis. By this I do not mean that every case should be operated upon when first seen, but that one should make up one's mind whether to follow a surgical or an exclusively medical plan. I am guided roughly by the amount of blood the patient has lost. If an effective operation is going to be performed, it will probably need to be a severe abdominal one, and an exsanguinated man is not in a state to stand it. Therefore, the hæmoglobin should be estimated, and when the patient has lost 50% of his blood, any surgery contemplated ought to be carried out without delay. Up to that point it is, perhaps, possible to bring about a cure by medical treatment, especially by blood transfusions, and it is advisable to try to do so. Sometimes it is possible to do big gastrectomies with a hæmoglobin estimation of 48%, even without blood transfusion, but when a man has had a sudden hæmorrhage he is in a precarious state.

Also I feel strongly that operation should never be performed during the collapse which immediately follows a big hæmorrhage; it is of no use to operate on a man who has suddenly lost a good deal of blood and whose pulse-rate is 110; he must be allowed from twelve to twenty-four hours to recover from the primary shock.

Lastly, as to the surgical procedure which may be decided upon. This should be the smallest possible, provided the bleeding point is ligated. Usually this means an excision, and the quickest partial gastrectomy for an ulcer of the lesser curvature is a sleeve resection. In the case of some of the ulcers which penetrate the pancreas, the quickest thing is to get into the lesser sac of the peritoneum and cut the ulcer

off the pancreas, leaving the base there, and then to invaginate the margins of the ulcer. At the time the operation is carried out the hæmorrhage has often stopped and a clot has formed in the vessel; if the ulcer surface is removed from contact with the gastric juice the bleeding is unlikely to be re-started. I have carried out this procedure successfully in the cases of two exsanguinated patients.

In dealing with the duodenum one is up against great difficulties, because excising a posterior ulcer in this situation is a long procedure, and I have only twice carried it out for hæmatemesis, a few hours after the onset—both times with success.

Dr. George Graham: I agree with Dr. Izod Bennett that these hæmatemesis patients, as a rule, recover if left without surgical attention. I intervene only to submit a point about the figures Dr. Aitken has mentioned. If one has to treat a person with severe hæmatemesis, it may be necessary to give not merely one transfusion, but two or three; one of my own patients had six transfusions. The difficulty the physician finds is in making up his mind as to whether the patient has been given enough blood in the first transfusion. Though he may be ill, his hæmoglobin may be 60% or even 80%, even after the loss of so much blood. The kind of case I am specially interested in is that in which the patient has lost much blood and the hæmoglobin has fallen to 40% or below. That means that the patient must be given blood. In some of these cases, on the day following the transfusion, the hæmoglobin reading was lower. It might be thought that this was because there had been more bleeding, but there was no good evidence of this. In one of my cases the drop was to 33%, in another to 29%. I believe it was because the patient was able to dilute the blood and restore the normal blood volume. The hæmoglobin reading should be taken before the transfusion and immediately afterwards, to ascertain how much the hæmoglobin had risen, and then should be repeated next day. After 600 c.c. of blood the hæmoglobin may rise from 38% to 50%; and when that figure is reached I leave the patient alone. But if the hæmoglobin either has not gone up or has gone down, the patient needs a second, possibly a third, transfusion. In the presence of a severe hæmatemesis, two or three transfusions greatly improve the general condition of the patient, and in many cases would save life.

Mr. Herbert Paterson said that in cases of acute hæmatemesis surgical interference was unnecessary, as with *adequate* medical treatment in very few was there a fatal issue. At a later period, when the patient had recovered from the results of the hæmorrhage, operation could be undertaken with much greater safety and with greater prospects of success.

Ten years ago he had given details of a series of 124 cases of severe hæmorrhage with no immediate deaths. Most of the patients had been under the joint care of either Dr. Soltau Fenwicke or Dr. Parkinson and himself. In most of these cases operation was performed at a later date, with a mortality rate of 2·5 per cent. At that time, Dr. Soltau Fenwicke stated that he had never seen a fatal instance of hæmorrhage from a gastric or duodenal ulcer. Since then he (the speaker) had not been so fortunate, as he had lost three patients from acute hæmatemesis. These three cases were of some interest. One was a case in which hæmorrhage occurred nearly ten years after gastro-jejunostomy for duodenal ulcer. The patient had remained quite well until he had a sudden severe hæmorrhage and died within forty-eight hours. At the autopsy no cause whatever for the bleeding could be discovered. Obviously, therefore, operation would have been futile. One of the other cases emphasized the importance of complete rest. This patient was admitted after two or three severe hæmorrhages, and with medical treatment did well for ten days. Then

one afternoon he reached out of bed to pick something off the floor, and shortly afterwards had a further hæmorrhage and died within a few hours.

These cases had not in the least shaken his belief that such cases were best treated medically. He had never operated earlier than two or three months after the hæmorrhage.

He considered that the outstanding need was absolute rest, not only physical rest but as complete rest as possible for the stomach. The teaching of Lenhartz, that patients could be fed on egg and milk immediately after a severe hæmatemesis, had done much harm. The stomach required rest, and nothing should be given by the mouth for at least three or four days. When he heard of high mortality rates in cases treated medically, he could not help wondering whether the medical treatment was really adequate and thorough. If the patients were kept *absolutely* at rest, physically and mentally, no food being given by the mouth, and repeated small doses of morphia and saline and glucose being administered by the rectum, the mortality rate was very low, far lower than any results obtained so far by surgical interference.

Some of the speakers appeared to suggest that in cases of hæmatemesis from an ulcer, operation should be performed immediately or within two or three days. This seemed to beg the question In any given case, how did one know that the bleeding was from an ulcer? Post-mortem observation indicated that this was a comparatively rare event. In the great majority of cases the bleeding was due to a general oozing from the gastric mucosa, and this was particularly the case in duodenal ulcer associated with high acidity.

He believed that surgical interference in cases of acute hæmatemesis was unnecessary, and rarely, if ever, justifiable.

Mr. Gordon-Taylor (in reply) said he agreed with Mr. Paterson as to the hæmorrhage sometimes following cases of suture of a perforated ulcer of the stomach or the duodenum. Apparently the operation had succeeded, then hæmorrhage occurred. They were calamitous cases, for the patients died, whether they were dealt with surgically or medically. On two occasions of the kind he had resected part of the stomach, and when sections were cut he found parenchymatous oozing over the whole of the gastric mucous membrane.

The Chairman said that the discussion had been concentrated on the best treatment of a patient with gastric or duodenal ulcer complicated by hæmatemesis.

There seemed to be general agreement that the indications for surgery were rare.

It had been emphasized that people suffering from hæmatemesis should not be moved, and that many of these patients who came to hospital ought not to have been sent. The fact remained, however, that many still did come, and a proportion—he did not know the exact proportion—died. From a survey of the cases which came to the post-mortem room it seemed clear that a small proportion might have been saved by operation.

The next point which had been agreed on was, that blood transfusion had helped both medical and surgical treatment in a certain number of cases. Blood transfusion, properly carried out and repeated, might bring patients back to a condition in which it was possible to operate, whereas before the transfusion, operation would have been fatal.

There was a difference of opinion as to the stage at which treatment should be undertaken. Most of the surgeons were emphatic in favour of operation—should operation be decided upon—after the first hæmorrhage, and not as one speaker had said, as the last resort.

Patients with hæmatemesis from gastric ulcer were suffering from secondary hæmorrhage, with which surgeons were familiar during the Great War. After two or more hæmorrhages the risk to life by any operation was increased, and therefore, if surgical treatment was contemplated it should be given early.

The next point arising was the character of the operation, should surgical treatment be performed. One important principle became clear, namely, that whatever the operation done, the surgeon must find the bleeding point and tie it.

There were a certain number of cases in regard to which it needed a nice judgment to determine the right treatment. Clearly there was no golden rule.